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PNEUMOCOCCUS ENDOCARDITIS*

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SATISFACTORY evidence of the pneumococcus origin of endocarditis entails the establishment of the organism in question as a cause of the process and its complete identification as a pneumococcus. The problem is complicated by the frequent occurrence of bacteriemia in lobar pneumonia as a manifestation of the disease and the possibility that pneumococci found in the blood or other organs are not the cause of the endocarditis. In the interest of strict accuracy, therefore, the pneumococcus should be demonstrated in the endocardial lesion itself as the bacterial incitant. It may be difficult or impossible, however, to obtain cultures uncontaminated by blood from small vegetations and reliance on identification of the organism in smears or sections by morphology and staining reaction alone is subject to the error of confusing the pneumococcus with certain strains of streptococci. Though compliance with the conditions will not always be possible, it may be said, that to establish the pneumococcus origin of an endocarditis, only organisms with the morphology and staining reaction of pneumococci should be demonstrated in smears or sections of the endocardial lesions, organisms with the growth peculiarities of the pneumococcus should be obtained in pure culture from the vegetations and complete identification should include an agglutination reaction with antipneumococcic serum or bile solubility or both. Further investigation of the character of the organisms found in acute and chronic pneumococcus endocarditis in man is desirable on account of the finding by Wadsworth (*Journ. Exp. Med.* 45, 1927) that the pneumococcus multiplying in the tissues of immunized horses becomes attenuated with loss of virulence, capacity to form capsules, susceptibility to phagocytosis and type-specificity.

The probable pneumococcus origin of endocarditis was established in 23 cases at the Massachusetts General Hospital. Of these 23 cases there are two in which lobar pneumonia was the apparent predisposing cause, but without other evidence of the bacterial incitant of the endocarditis than the finding of organisms with the morphology and staining reaction of pneumococci in pericardial fluid in one and in empyema fluid in another. There are twelve additional postpneumonic cases, four with pneumococcic meningitis. Of the twelve, pneumococci were found by blood culture during life in five, and the heart's blood after death in nine, and microscopic examination of smears or sections of the vegetations showed organisms with the morphology and staining reaction of pneumococci in six. In a thirteenth case, bronchopneumonia preceded the endocarditis. In this case both pneumococci and streptococci were found by culture from the heart's blood and organisms resembling pneumococci in smears from the vegetations. In five additional cases, there was no history of pneumonia but the finding of pneumococcus septicemia by culture of the heart's blood in four and the spleen in one offered presumptive evidence of the pneumococcus origin of the endocarditis. In the three remaining cases there was a pneumococcic meningitis and pneumococci were demonstrated by blood culture during life, in culture from the heart's blood after death or by culture from the spleen. In only four instances in the series was the type of pneumococcus determined. Type I was recovered by blood culture during life in two. Type II was identified in the sputum during the pneumonia in one. Type III was found in the pneumonic lung in one.

The frequency with which acute endocarditis occurs during life in patients with lobar pneumonia is uncertain on account of the difficulty of establishing the diagnosis. Acute endocarditis at autopsy in patients with or after recovery from lobar pneumonia at the Massachusetts General was found in 14 (4.15%) of 337 cases, from March, 1898 to February, 1929. Lobar pneumonia is the most common predisposing cause of pneumococcus endocarditis. Bronchopneumonia may also be responsible. The portal of entry is uncertain in about a third of our cases.

The sexes are about equally represented. Approximately one-half the cases occurred during the fourth, fifth and sixth decades of life. Preëxisting valvular disease is observed less often with pneumococcus than with streptococcus en-

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docarditis. In 8 of the 23 cases, chronic as well as acute endocarditis was present.

Pneumococcus endocarditis may end fatally in spite of the presence of a considerable degree of active immunity. In one case of the series, empyema followed pneumonia and failed to improve after surgical drainage. On the eighty-third day of the disease the blood culture was positive for Type I pneumococcus in spite of the presence of immune substances in the blood sufficient to protect mice against 100,000 lethal doses of the homologous organism. Autopsy three days later showed endocarditis. Microscopic examination of the vegetation showed numerous cocci and diplococci.

As with other types of valvular lesions, the pneumococcus infection is usually confined to the left side of the heart, as in 18 of the 23 cases. The right side was alone infected in three cases and both sides were involved in two. The mitral valve was affected in 13 instances, the aortic in 12 and both in five. The tricuspid valve was involved in five, including one in which the pulmonary valve was also affected.

The vegetations vary from minute to large masses of a grayish or yellowish color. They are soft and friable or dense. Both proliferative and ulcerative types occur.

The symptoms include remittent or intermittent fever, chills and sweats, anemia and leucocytosis. Joint pains or arthritis and clubbing of the fingers may occur. Purpuric spots are found in a small proportion of the cases. They were noted in four instances in the series. Significant findings on examination of the heart are usually lacking. Harsh, loud, or musical, systolic murmurs may be regarded as significant when they develop under observation. Diastolic murmurs are also important, unless they antedate the present illness.

The course of the disease is usually short and terminates fatally within a few days to a few weeks. The duration is difficult to estimate on account of the difficulty of fixing the time of onset. Meningitis is a frequent complication and was observed in 7 of 23 cases.

Pneumococcus endocarditis may be suspected when fever, not otherwise to be explained, continues after subsidence of a pneumonia. The occurrence of purpuric spots, arthritic pains and clubbing of the fingers is still further suggestive. The development under observation in such cases of diastolic or loud, harsh or musical, systolic murmurs goes far toward establishing the presence of a valvular lesion and the finding of pneumococci in the blood stream leaves little room for doubt as to the presence of a pneumococcus endocarditis.

Though it is probable that pneumococcus endocarditis is not inevitably fatal, I know of no instances in which the diagnoses have been adequately established and recovery has taken place.

The chief measure of importance in the prevention of pneumococcus endocarditis is, of course, the prevention of pneumococcus pneumonia and our efforts in this direction are, thus far, almost wholly unsuccessful. It is, however, to be expected that early specific serum treatment of pneumonia with resulting elimination of pneumococci from the blood stream will diminish the incidence of pneumococcus endocarditis as a complication.

There are no special indications regarding treatment of pneumococcus endocarditis other than the use of homologous antipneumococcic serum, with the usual precautions to guard against reactions in individuals who may be sensitive to horse serum. The problem may be complicated by the recent administration of serum in the treatment of pneumonia and consequent hypersensitiveness. Special caution should be exercised in such cases. The presence of serum disease or a conjunctival reaction to horse serum contraindicates the use of the antipneumococcic serum.

ACUTE BACTERIAL ENDOCARDITIS*

BY CADIS PHIPPS, M.D.†

THE simple definition of endocarditis, namely an inflammation of the endocardium, should perhaps be borne in mind in discussing acute bacterial endocarditis lest we confuse it with other conditions (such as sepsis), which lack actual endocardial infection. Acute bacterial endocarditis is of course closely related to sepsis and probably one-fifth of all cases of sepsis are accompanied by bacterial invasion of the valves. Its clinical incidence in relation to other forms of heart disease is now more clearly recognized and is perhaps worthy of a minute's consideration. It is because of the improvement in laboratory technique (such as blood culture), especially during the last decade that we get our best data from the more recent case reports and furthermore, from a study of the older records, where the term, "Malignant Endocarditis" was the common one, it would seem that the incidence of bacterial endocarditis was increasing.

For the fifteen years prior to 1929, I obtained from records at the Boston City Hospital, clinical reference to only 188 cases, of which 21 were acute and although I shall not use them for statistical purposes in this paper, the following figures, based on clinical records, are of

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some interest. Cases treated in the First and Third Medical Services at the Boston City Hospital during the year 1929 were 3305; cardiac cases 711; bacterial endocarditis 33; acute bacterial endocarditis 4. Because of the difficulty and possible error, however, in antemortem diagnoses in this condition, I am depending on cases which were autopsied (most of the records being furnished me through the courtesy of Dr. Mallory), for the incidence of the different organisms and other statistical data. Also, I am of course restricting myself to *acute* bacterial endocarditis and omitting pneumococcus endocarditis.

Acute bacterial endocarditis is in general a fulminating process with a rapidly fatal outcome and although in many instances the cardiac manifestations may be the most striking, yet essentially the picture is largely one of an acute sepsis modified by the underlying condition or exciting cause. This fact compared with the symptomatology of subacute bacterial endocarditis suggests bacteria of a greater virulence either in general toxicity or, if sufficient time elapses, in the power to produce necrosis of valve tissue ("ulcerative endocarditis"). While, as I have said, the symptoms are practically those of a rapidly fatal septicemia, yet in certain instances it is difficult to determine the exact time at which the endocardial process began, and in others the bacterial virulence may be of a lesser degree and the progress of the disease approach more the picture of subacute bacterial endocarditis. Furthermore, the type of invading organism and more particularly the underlying conditions may alter the clinical course and for that reason I shall first of all mention the different varieties.

Total number of cases—44

(1) Streptococcus Hemolyticus	21 cases
(2) Staphylococcus Aureus	11 cases
(3) B. Coli Communis	5 cases
(4) Gonococcus	2 cases
(5) Meningococcus	2 cases
(6) Staphylococcus Albus	1 case
and also a combination of Streptococcus Hemolyticus and Staphylococcus Aureus	1 case and
B. Coli Communis and Staphylococcus Aureus	1 case

(1) The 21 cases due to streptococcus hemolyticus included 17 males and four females with ages ranging from 11 to 74 years. The commonest age (not the average) was 45 to 50 years. In about 60% of the cases there was evidence of old pathology in the endocardium, particularly arteriosclerotic changes. As regards the valves affected, nine cases showed involvement of the mitral, three of the aortic, eight of both mitral and aortic and one of the tricuspid. The duration of the disease varied from four days to three weeks (one case of nine weeks). The exciting causes were roughly the following: respiratory infections in nine instances; surgery in five; erysipelas in three; intestinal ulceration in one and probably diseased teeth and tonsils in the others. The symptoms were essentially those of the underlying cause with a more or less abrupt change in the temperature and pulse curves, the appearance of new murmurs and in about a third of the cases some petechial spots. The antemortem diagnosis when made was essentially by means of a positive blood culture.

(2) Staphylococcus, 11 cases; males nine and females two; age incidence two to 70 years (especially 45-50 years); valves affected, mitral five, aortic three, mitral and aortic two, mitral and aortic and tricuspid one.

Exciting Causes: five surgical, one postpartum, one lung abscess, two intestinal ulceration, two teeth and arteriosclerosis; duration two days to three weeks. Of course a striking feature of acute bacterial endocarditis due to this organism is the pyemic tendency. The following case is illustrative: A fifteen year old boy received a contusion of the left knee one week before entrance to the hospital and two days after admission, he died, a blood culture being positive for staphylococcus aureus. Postmortem examination showed septic arthritis of left knee; osteomyelitis of left tibia, mitral endocarditis, purulent pericarditis, bronchopneumonia and multiple abscesses in the liver, lungs, etc.

(3) B. Coli Communis five cases; males three and females two; ages 13, 15, 61, 68, 75. Valves affected, mitral two, aortic and mitral one, tricuspid (only) one, all three valves one, duration five days to two weeks. All patients apparently were debilitated; in one case there had been a laparotomy. Symptoms were those of the preëxisting disease until the onset of a septicemia and the organisms were found in blood cultures.

(4) Gonococcus two cases; two females 21 and 26 years; one postpartum, one pus tube, duration one week. Thayer says the aortic valve is the chief one to suffer; in these cases one showed invasion of the aortic valve, the other the mitral.

(5) Meningococcus two cases; one male and one female; 21 years and 20 years; valves, first, aortic (on an old lesion), secondly, mitral. The symptoms were those of meningitis and the endocarditis was not diagnosed until autopsy. This form of endocarditis has been considered rare, but it has been shown (Veillon[1]) that it may exist extensively without cardiac murmurs while on the other hand several have spoken of murmurs appearing during a meningitis and disappearing under the administration of serum. Arthur M. Master[2] a year ago described three cases of meningococcemia with definite signs of valvular involvement, the cardiac signs apparently being removed by serum: the cases, however,

were essentially subacute and not within the scope of this paper.

(6) The one case of staphylococcus albus was in a girl of 17, the onset acute and with no causal pathology noted except abscessed teeth. The patient lived about a week. The mitral valve was the one affected.

(7) The case showing both streptococcus hemolyticus and staphylococcus aureus occurred in a male of 31, ill with pneumonia, and it involved the mitral valve and the patient lived three weeks.

(8) The last one, a combination of staphylococcus aureus and B. coli communis was in a male of 45 and coincident with intestinal ulceration. The mitral valve was affected and the duration was three weeks.

The pathology I have not attempted to discuss.

The diagnosis of acute bacterial endocarditis will rest therefore upon a picture of sepsis, often arising in the midst of some acute or chronic affection with appearance of murmurs, often in addition to preëxisting ones; it will be materially strengthened by successful blood cultures, although we must remember that a negative one does not exclude the possibility, nor does a positive one prove that a valve is affected. The only 100 per cent sure diagnostic method I believe, is an autopsy. This last statement should perhaps be considered in relation to opinions concerning prognosis and treatment.

The prognosis is practically always fatal. Recoveries have been reported and it would seem fair to believe that this might be possible in meningococcic endocarditis where we have a specific serum. Many attempts have been made at specific treatment other than with serum. Gentian violet and mercurochrome, while not so illogical in this condition as they are in subacute bacterial endocarditis, have not proved of real value in my mind. Glucose solution given intravenously, a common therapy at present, while it did help one patient temporarily, probably because he had an underlying arteriosclerotic heart, has been followed in three other cases seen by me, by a definite increase in the severity of the symptoms.

About ten years ago I believed that a blood transfusion might be a valuable means of treatment in cases of acute bacterial endocarditis, due to staphylococcus aureus because of the relief which was apparently obtained in two possible cases. Both showed positive blood cultures, developed definite signs of endocarditis, and when the prognosis was practically hopeless, made sudden and marked improvement with eventual complete recovery following blood transfusion. I have not seen a blood transfusion give similar relief in any other cases, however.

CONCLUSIONS: Forty-four cases of acute bacterial endocarditis have been offered. The sex incidence is 32 males and 12 females and the age incidence in general 45-50 years. Streptococcus hemolyticus was responsible for about 50 per cent and staphylococcus aureus for 25 per cent. The duration was roughly from three days to three weeks. The left heart was by far the commoner to become involved and ordinarily there was a preëxisting valve lesion (but so far removed from rheumatic fever as to make the question of allergy highly improbable). The treatment is almost entirely symptomatic (digitalis, salicylates, etc.,) and apparent recovery of some cases rather doubtful, although in the case of meningococcus this seems quite possible.

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SUBACUTE STREPTOCOCCUS ENDOCARDITIS*

BY HYMAN MORRISON, M.D.†

MY part of the program deals with the subacute form of bacterial endocarditis, as interesting and just as serious a disease entity as the acute form, and more common. Thanks to the intensive studies during the past quarter of a century by investigators both here and abroad—subacute bacterial endocarditis is now well understood from the clinical, pathological, and bacteriological points of view. Still it presents several major problems,—among them the question of its relationship to rheumatic fever, the futility to date of all therapeutic effort, and the paradoxical situation of an organism of low virulence, the anhemolytic streptococcus, giving rise to an infection which is practically always fatal. At least from the laboratory point of view the patient is immune against this organism, for his blood serum shows a high agglutinin titer against it; nevertheless it flourishes in enormous numbers on the very surface of the vegetations. Howell and Corrigan and also Swift found that patients with subacute endocarditis do not show skin reactions to filtrates of the streptococci recovered from their blood, and Kinsella and Garcia were unable to induce any local reaction by inoculat-

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ing patients ill with this disease with living streptococci. Experimentally, investigators (Kinsella, Wright, Wordsworth, and White) were able to produce bacterial endocarditis regularly on traumatized valves in animals only if they were previously immunized. In contrast to this Homer Swift and his coworkers pointed out that patients with rheumatic fever, a much milder infection, do not show such a serum immunity against the strains of streptococci occasionally recovered from them. Apropos this relationship he cites the case of a patient under his care who entered the hospital with rheumatic fever and during the next two months had all of the exudative phenomena usually seen, with pericarditis, pleurisy, and polyarthritis. He then seemed to recover, but after an acute sinusitis developed classical signs of subacute bacterial endocarditis, and showed the streptococcus viridans repeatedly in blood cultures. His blood which had been saved from the period when he was suffering from rheumatic fever showed no agglutinins, while that obtained after the development of subacute bacterial endocarditis showed them in a fairly high titer.

How, in subacute bacterial endocarditis, do organisms invade the blood stream and colonize on the cardiac valves of an immune individual? Is there involved another primary causative agent, a virus still unknown which, as is thought to be the mechanism in influenza, prepares the soil for the one that is known? Or is there involved the factor of a special selective affinity on the part of the anhemolytic streptococcus for the endocardium, changing an organism ordinarily low in virulence to a high degree of vitality? A reverse action is seen in the case of the meningococcus, highly virulent when it invades the central nervous system, but becoming relatively less virulent when it implants itself on the endocardium. Or is it as Swift holds that subacute bacterial endocarditis is accompanied by a state of relative tissue immunity and that the difference between the man ill with rheumatic fever and the one with subacute bacterial endocarditis rests in altered tissue reactivity and that bacterial localization on the endocardium results from its altered capacity to react toward bacteria?

INCIDENCE AND ETIOLOGY

In 1927 I reported on the incidence of subacute bacterial endocarditis at the Massachusetts General Hospital between 1904 and 1926 inclusive. Up to 1922 the number of patients with this disease ranged between one or two to six or eight cases per year. In the decade that followed, its incidence at this Hospital more than doubled. The accompanying curve gives a graphic representation of these figures. Undoubtedly this increase is due chiefly to the better understanding of this disease and also to the more general use of blood culturing as a diagnostic method. At least in this part of the country it has become pretty well recognized that protracted fever in individuals who have chronic valvular disease must raise the suspicion of subacute bacterial endocarditis.

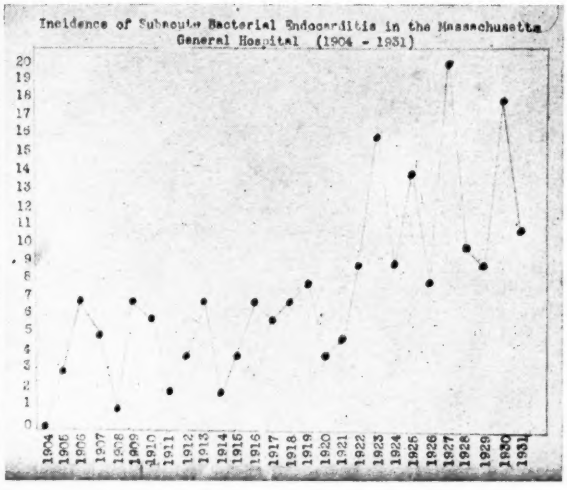

Table I gives a comparative analysis of cases of rheumatic fever and of subacute bacterial endocarditis admitted to six of the hospitals in Boston during the past four years. A point of minor interest is that the ratio between the number of cases of subacute bacterial endocarditis and that of rheumatic fever admitted to all these hospitals, except the Children's, is about one to three. I do not mean to imply by this that a third of the cases of rheumatic fever eventually end up in subacute bacterial endocarditis. The ratio is merely a comparison between the cases that enter hospitals, and it is clear, of course, that many cases of rheumatic fever never leave home, while most individuals with subacute bacterial endocarditis sooner or later go through a residence in a hospital. It, however, does emphasize that persons who have had rheumatic fever are the potential material for subacute bacterial endocarditis. Actually according to White and Jones, here in New England, it occurs in one out of every twenty-five cases of rheumatic fever.

Another point which this table brings out is that the Children's Hospital has had the least number of cases of this disease. This substantiates the known fact that subacute bacterial endocarditis is prevalent in young adult and in early middle life, following the period of highest incidence of rheumatic fever with prolific valvular damage. This point will be referred to again in discussing the etiological relationship of subacute bacterial endocarditis to rheumatic fever.

BACTERIOLOGY

In his brilliant contributions to this disease entity Emanuel Libman suggested that the cases of bacterial endocarditis be designated according to the course and the infecting organism. Because the streptococcus, and practically always its anhemolytic strains, is the causative agent in such a large number of the subacute form he designated it subacute streptococcus endocarditis. This name has not taken hold, however, rather has the term subacute bacterial endocarditis acquired the implication that the non-hemolytic streptococcus is the infecting organism, unless another, such as the influenza bacillus, is definitely mentioned. Though Libman states that the non-hemolytic streptococcus is the organism which causes this disease, still in this present study there were typical cases which were distinctly due to other bacteria, namely the hemolytic streptococcus, the staphylococcus, and a diphtheroid bacillus. The literature confirms this, and records the gonococcus, the pneumococcus, and the meningococcus as infecting bacteria. This also will be referred to again.

TABLE I

ADMISSIONS OF CASES OF RHEUMATIC FEVER AND SUBACUTE BACTERIAL ENDOCARDITIS IN BOSTON HOSPITALS 1928-1931

	Rheumatic Fever					Subacute Bacterial Endocarditis					Ratio of S. B. E. to R. F.
	1928	1929	1930	1931	Total	1928	1929	1930	1931	Total	
Beth Israel	4	24	22	21	71	3	4	10	3	20	0.3—
Boston City	138	145	—	—	283	32	22	—	—	54	.2—
Children's	27	38	29	28	122	2	1	1	1	5	0.04
Mass. Gen.	35	45	44	38	162	10	9	18	11	48	0.3—
Mass. Mem.	2	7	15	4	28	1	1	4	4	10	0.3+
P. B. Brigham	14	38	31	23	106	7	10	5	18	40	0.3+

PATHOLOGY

Certain pathological aspects peculiar to subacute bacterial endocarditis make it stand out clearly in contrast to other infectious diseases involving the heart. It is essentially an endocarditis, rarely involving the myocardium and pericardium so common in rheumatic fever, and rarely attended by exudative phenomena. Clinically this accounts for the rarity of the arrhythmias, pericarditis, pleurisy, and polyarthritis in this disease; also for the rarity of electrocardiographic evidence of parenchymatous irritation of the heart as indicated in

TABLE II

BLOOD CULTURE EXAMINATION IN SUBACUTE BACTERIAL ENDOCARDITIS IN BOSTON HOSPITALS

	Streptococcus				Staphylococcus		Diphtheroid Bacillus	No Growth	Total Number of Cases Cultured
	Viridans	Non-hem.	Hemolytic	Atypical	Aureus	Albus			
Beth Israel	8	4	1	2	1	1	1	2	20
Boston City	14		1	3	—	1	—	8	27
Mass. Gen.	22	1	—	—	1	—	2	11	37
Peter Bent Brigham	69	1	—	2	—	—	—	28	100
Total	113	6	2	7	2	2	3	49	184

Table II summarizes blood culture examination in this disease in four of the Boston hospitals. Libman succeeded in growing anhemolytic streptococci in the blood of 95 per cent of his cases. If to the cases in this series showing the presence of the anhemolytic streptococcus are added those with atypical strains and also those with negative cultures, which likely were positive at some time, then the non-hemolytic streptococcus is responsible for 95 per cent of the cases; other organisms such as the staphylococcus, hemolytic streptococcus and diphtheroid bacilli accounting for the remaining 5 per cent. It must be borne in mind that in suspected cases blood culture examinations must be repeated and repeated—that a growth in the culture may not appear till after a week or more. On the other hand, at times the streptococcus viridans may be found in the blood of patients suffering from other diseases than endocarditis, and rarely in normal individuals.

lengthened P-R interval and alterations in the QRST complex. It is always secondary, occurring on previously damaged valves, most commonly of rheumatic origin, but occasionally of luetic or arteriosclerotic or congenital origin. The data on this point available in 203 cases in four Boston hospitals are classified in Table III showing that subacute bacterial endocarditis was superimposed on old rheumatic valvulitis

in at least 68 per cent of the cases, and very likely in more, for of the 54 in which there was no history of acute infection a good proportion must have had rheumatic fever or an allied infection in some form at some time.

TABLE III

ETIOLOGY OF PRIMARY ENDOCARDITIS ACCORDING TO HISTORY

	Total Number	Rheumatic Fever and Allied Infections	No History of Rheumatic Fever	Congenital	Luetic	Arterio-sclerotic
Beth Israel	20	15	2	3	0	0
Children's	35	22	13	—	—	—
Mass. Gen.	37	31	0	3	2	1
P. B. Brigham	111	66	39	5	1	—
Total	203	134	54	11	3	1

Another well-recognized, interesting fact emphasized by postmortem (Table IV) data on this disease in the same hospitals is that in 80 out of 88 cases the valves involved were of the left side of the heart. The same relation to the left side of the heart also holds true in rheumatic endocarditis; this may partly be explained by the greater vascularization of the valves of the left side of the heart in adult life. It involves more frequently the mitral than the aortic, but even more frequently both these valves together. The congenital basis for the process is most commonly a bicuspid aortic valve or a patent ductus arteriosus;—one of the Beth Israel cases involved an interventricular septal defect. Frequently in this disease, as also in the acute bacterial type and uncommonly in rheumatic fever, there is involvement of the mural endocardium—auricle and ventricle and chordae tendineae. S. A. Levine and H. B. Sprague, in studies correlating clinical evidence of valvular deformity with postmortem findings, stress the fact that it is unusual to find subacute bacterial endocarditis on a mitral valve with well-marked stenosis.

Rheumatic fever is characterized by small, uniform verrucous or warty nodules—resulting from a swelling and desquamation of the endothelial cells at the point of injury; the denuded area is then covered with agglutinated blood platelets; the adjacent endothelium proliferates and covers the surface of this nodule, thus limiting its size. Later the nodule becomes organized and flattened, firm and smooth leading to scarring along the line of closure of the valve and resulting in valvular insufficiency and stenosis. In contrast, subacute bacterial endocarditis is characterized by larger lesions with round cell infiltration, the agglutinated blood platelet-mass is invaded by large numbers of bacteria, which inhibit the spread of the endothelium of the adjacent endocardium over its surface,—the process then spreads by continuity. The freshly formed superficial portions of this vegetation consist almost entirely of bacteria; in the deeper portion, the bacteria tend to diminish, fibroblasts appear, and, when there is healing, connective tissue is formed. These vegetations vary in size and shape, are greenish, reddish or yellowish in color, and are soft and friable in consistency. They are not limited to the line of closure of the valve as in rheumatic fever, but spread over their surfaces and also over the endocardium of the auricles and ventricles and over the chordae tendineae. There is little tendency to ulceration as contrasted with the acute bacterial type. It is because these vegetations are large, soft, and friable and uncovered that particles can break loose into the blood stream and produce the embolic phenomena so commonly found in subacute bacterial endocarditis.

TABLE IV

VALVES INVOLVED IN SUBACUTE BACTERIAL ENDOCARDITIS ACCORDING TO POSTMORTEM EXAMINATIONS IN BOSTON HOSPITALS

	Mitral	Aortic	Mitral + Aortic	Mitral + Aortic + Tricuspid	All Valves	Pulm. + Aortic	Tricus. + Vent. Sept. Defect	Pulmonary and Duct. Art.	Pulm., Aortic Duct. Art.	Only Mural Endocardium	Total
Boston City	14	8	20	2	1	2	—	—	—	—	47
Beth Israel	3	—	2	—	—	—	1	—	—	—	6
Mass. Gen.	1	1	3	—	—	—	—	1	—	1	7
P. B. Brigham	7	9	13	—	—	—	—	—	1	—	30
Total	25	18	38	2	1	2	1	1	1	1	90

SYMPTOMATOLOGY AND CLINICAL COURSE

In the light of this pathological background and of the well-known low virulence of the anhemolytic streptococcus the symptomatology of subacute bacterial endocarditis becomes unusually clear. Its clinical course is primarily that of a low grade sepsis modified ultimately by a variety of signs and symptoms due to multiple embolism. The onset is almost always insidious with general malaise; occasionally the disease first attracts attention by an acute embolic accident, such as abdominal pain. There is progressive secondary anemia giving the café-au-lait facies, with progressive loss of strength and weight, and clubbing of fingers. The pulse may be rapid from the first and dyspnea is a common feature. The fever is rather low at the outset, but takes on the septic swing with chills and sweats as the embolic phenomena manifest themselves in the important organs.

Multiple embolism manifests itself in petechiae in skin, tissues, mucous and epithelial surfaces, and in the fundi, in infarctions of the thoracic and abdominal organs and of the brain and the extremities, in embolic or mycotic aneurysms, and in focal embolic lesions in the kidney. Any artery may be affected by embolic aneurysm—most commonly those of the brain; when such an aneurysm ruptures, subarachnoid or intraventricular hemorrhage may result, giving rise to a syndrome simulating meningitis. Splenomegaly is due both to sepsis and to infarction. Embolism in the kidney may give rise to petechial hemorrhages, to infarction, and to focal glomerular nephritis; the latter is not seen in any other type of endocarditis, the petechial, spotted appearance gives the term of "flea-bitten kidney". Absence of suppuration in this disease is a striking feature.

Clinical laboratory examinations show the leucocyte count at first low, then rising to a high level with the more severe embolic lesions; histiocytes may appear in the peripheral circulation. The progressive anemia is due to sepsis, petechial hemorrhages, and at times to nephritis. The urine almost always shows red blood corpuscles; with renal infarction there may be gross hematuria; and with embolic glomerulonephritis there is a picture of acute nephritis. With the latter there may be edema and uremia. Death is generally due to sepsis after a duration of from three to twenty-four months, average six months. Cardiac congestive failure and embolism are contributing causes, and occasionally pneumonia or uremia.

THE BACTERIA-FREE STAGE

Libman draws distinction between bacterial and bacteria-free stages in this disease; the former represents the active phase—bacteria are found in large numbers in the vegetations and in the bloodstream. If the patient does not die of the bacterial infection he may pass into the bacteria-free stage, especially if the infection itself has been comparatively mild. In this stage the disease is no longer active and bacteria cannot be demonstrated in the valvular lesions;—the latter show evidence of healing in the shape of fibrosis, or calcification—also fibrosis in the embolic glomerular lesions of the kidneys. The patient dies not of infection, but of sequelae such as cardiac failure, anemia and uremia.

Libman believes that the milder forms of this disease may go unrecognized, and that probably many cases recover spontaneously. Oille, Graham and Detweiler reported twenty-three cases in Toronto in 1915, all of which recovered. These patients had slight fever and murmurs; some had petechiae and splenomegaly; in all, the streptococcus viridans was isolated from the blood. A similar group of eighteen cases in Prague in 1920 was reported by Sulus. Libman himself has reported ten typical cases completely recovered.

TREATMENT

Treatment in this disease has thus far been futile. To begin with, the disease is always superimposed on chronic endocarditis. Furthermore the nature of the endocardial lesion is such as to produce multiple embolism. Thirdly, the immunological aspect of the disease is not yet understood. Because the etiological basis for this disease is so frequently rheumatic fever, the problem resolves itself to one of prevention of the latter disease. A year ago I felt that it would be a worthwhile effort to vaccinate with streptococcus viridans people who had rheumatic valvular disease but who were symptom-free, the very people who fall victims to subacute bacterial endocarditis; preferably with the predominant streptococcus cultured from their throats. Clinically it seemed plausible, and subsequently I learned that Libman had recommended such vaccination on a large scale about six years ago. However, it was pointed out then that it is the rheumatic fever patient who shows an immune serum reaction to streptococci that is most likely to develop subacute bacterial endocarditis. Drs. Hans Zinsser and Benjamin White thought vaccination impracticable. The streptococcus is very illusive as to strain and it is known that the immunity which it confers is short-lived and very uncertain, and furthermore, as was pointed out before, the patients with this disease already show a high degree of immunity against the organism recovered from the blood stream.

RHEUMATIC FEVER AND SUBACUTE BACTERIAL ENDOCARDITIS

The relationship between rheumatic fever and subacute bacterial endocarditis is still a matter of difference of opinion. There are those who believe that the two are but different phases

of the same process; one the mild and the other the severe form of the same infection; that the streptococcus viridans may sometimes be found in the blood stream of patients ill with rheumatic fever as well as in subacute bacterial endocarditis; that experimentally the injection of anhemolytic streptococci in a series of animals will produce a progressively destructive type of lesion in some and a simple healing type in others; that the transition between one and the other is gradual, it being hard to tell where one ends and the other begins; that the two types of vegetation may be found at the same time in the same valve or even in the same cusp; and that there is no fundamental difference in the histology of the vegetations.

On the other hand it is held that the two are independent disease entities and that the only common factor is that the valvular damage done by rheumatic fever provides most commonly the basis on which bacteria are planted. However, lues, arteriosclerosis, and a congenital defect may be the basis for subacute bacterial endocarditis. Again though the anhemolytic strain is the most frequent infectious agent in this disease, still typical instances are met in which the hemolytic strain, the staphylococcus, the pneumococcus, the meningococcus or diphtheroid bacilli are positive organisms. It has also been noted that subacute bacterial endocarditis occurs rarely in children, the age group in which rheumatic fever is prevalent; and if it does it is on a congenital basis chiefly. Finally, because the concurrence of the two apparently independent disease processes has been noted in the same individual it seems unjustifiable to consider subacute bacterial endocarditis as a phase of rheumatic fever with a common etiology and different only in tissue response.

It is within the memory of many of us that pulmonary tuberculosis was considered practically a hopeless disease. Recently I was consulted by a young woman ill with a low grade fever of several weeks' duration. Because she had lost a sister with tuberculosis she was in dread that she, too, had this disease and hoped her illness was anything but consumption. Examination, however, revealed old rheumatic heart disease. This was substantiated by the history. In my notes I commented that I wished that she did have tuberculosis rather than what I suspected. This change of attitude among us to tuberculosis merely emphasizes that there should be no room for pessimism in medicine; that what seems hopeless today will, if human ingenuity has free play, take on new hope tomorrow.

MORTALITY STATISTICS IN THE DEATH REGISTRATION AREA OF CONTINENTAL UNITED STATES: 1931

Washington, D. C., October 12, 1932. — The Department of Commerce announces that in 1931 in the death registration area of continental United States (exclusive of the State of Utah) there were 1,318,111 deaths representing a mortality rate of 11.1 per 1,000 estimated population. This is the lowest rate since the Bureau began the annual collection of mortality statistics in 1900. It is estimated that 96.3 per cent of the total population of the United States was included in the registration area for the year 1931. The figures for the State of Utah are not included due to the failure of that state to furnish the Bureau with any certificates of death for the year 1931. However, even if the number of deaths which occurred in that state were included they would not change the death rate for the year 1931, so that the rates for that year may be considered as comparable with those of the three prior years shown in the report.

In the registration area as a whole and in the majority of the individual states the death rate was higher for the urban area than for the rural. The exceptions to this statement were California, Illinois, Indiana, Massachusetts, Michigan, New Hampshire, New Jersey, and New York, where the rates for rural areas were higher than those for urban areas, and Wisconsin, where the urban and rural rates were the same.

In the general decrease in the death rate, a rate has been reached in some states that may be considered remarkable. In 11 states the number of certificates filed indicates a death rate of 10 or less. In fact, four states, North Dakota, South Dakota, Oklahoma, and Wyoming, have rates of less than 9 per 1,000 estimated population.

Exceptionally high mortality rates may indicate that the area is a hospital zone to which many nonresidents from the surrounding region are brought for treatment. In the case of very low rates the reverse may be true, namely, that the area is entirely residential in character, without hospital facilities, often situated near a large city in which the residents are treated for illness. No attempt has been made to allocate the certificates to the places of residence of the decedent.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending October 15, indicate a mortality rate of 10.2 as against a rate of 10.0 for the corresponding week of last year. The highest rate (18.5) appears for Trenton, N. J., and the lowest (4.2) for South Bend, Ind. The highest infant mortality rate (15.8) appears for Wilmington, Del., and the lowest for Bridgeport, Conn., Fort Wayne, Ind., Lynn, Mass., New Bedford, Mass., Peoria, Ill., and Youngstown, Ohio, which reported no infant mortality.

The annual rate for 85 cities is 11.1 for the forty-one weeks of 1932, as against a rate of 11.9 for the corresponding period of the previous year.—*Bulletin of the Census.*

ACUTE MONOCYTIC LEUKEMIA

A Case Report

BY STUART N. GARDNER, M.D.*

AMONG the unusual types of leukemia is monocytic leukemia, believed by many to be distinct from the two more common types, lymphatic and myelogenous. This third type, arising from the reticulo-endothelial (histiocytic) system and characterized hematologically by an extreme degree of monocytosis and histiocytosis, was first described by Reschad and Schilling-Torgau in 1913. Since then several other writers, including Dameshek[1], have described and reported cases of this condition, bringing the total number of such cases up to twenty-nine. Some of these, however, were rejected by Dameshek after careful analysis on the grounds that they were definitely myeloblastic or myelocytic leukemia or could not be differentiated from agranulocytosis. The total number of well-substantiated, fully described cases, was placed at eighteen, including his two cases. More recently four more cases have been reported and another such case is described in this paper.

CASE REPORT. Mrs. M. F. C. (Salem Hospital, 57118), aged 47 years, was admitted to the medical service of the hospital, November 23, 1931, complaining of dyspnea of two days' duration.

HISTORY. Four days before admission she felt weak, had a sore throat and consulted her physician who found the posterior cervical glands to be swollen. She had a temperature varying between 100.5°F. and 101.5°F. She had complained of no cough, but on the day prior to admission she began to have pain in her right chest, originating below the scapula and aggravated by deep breathing. She had had a hysterectomy for a uterine fibroid in 1929. The first child was still-born; she had one son, aged 17 years, living and well; since then she had one miscarriage. Otherwise, the family and marital history were not significant.

EXAMINATION showed an alert white woman, flushed and in some respiratory distress. The tonsils were swollen. The left lung showed crepitant râles at the base extending toward the mid-axilla. The heart sounds were regular but weak; blood pressure was 100/80. There was slight abdominal distention. The temperature was 100.4°F.; pulse, 120; respiration, 56. Urine examination showed a slight trace of albumin, and in the sediment 8 to 10 free white blood cells, with a few small clumps, and a few finely granular casts. Blood examination showed red cells, 3,150,000; hemoglobin, 65 per cent; leukocytes, 21,500; neutrophils, 69 per cent; lymphocytes, 25 per cent; monocytes 6 per cent. The Wassermann test was negative. Roentgen ray examination showed no intrathoracic pathology.

CLINICAL COURSE AND SUBSEQUENT EXAMINATIONS. Her condition gradually improved except that she was apprehensive and continued to have night sweats. Her temperature, pulse and respirations gradually fell. Another Roentgen ray examination on December 1 showed no change. On this date many spirochetes and fusiform bacilli were found in the sputum but no acid fast bacilli. The white count was 17,800 with a differential of neutrophils, 68 per cent; lymphocytes, 17 per cent; monocytes, 13 per cent; eosinophils, two per cent. On December 3, the white count was 21,900 with the differential showing again an increase in monocytes, 16 per cent. She was allowed to sit up on December 7. On December 9, some pressure blebs on her buttocks were incised. On December 14, her left ankle became slightly edematous and December 19, she complained of sore throat. The faucial pillars were edematous and reddened and the right anterior cervical glands were enlarged. A smear from the throat was negative for Klebs-Loeffler bacilli but showed staphylococcus aureus. At this time she began to complain of gingivitis. A smear from the gums showed many spirochetes with only a rare fusiform bacillus. A dental consultant recommended sodium perborate as a mouth wash and argyrol applied to the gums. On the following day she was seen by a throat consultant who suggested Vincent's angina as a possibility and in view of marked swelling and redness of the right tonsil, suggested a peritonsillar abscess. In the meantime her temperature and pulse had been rising until the former was 100°F. in the morning and 102°F. in the afternoon with a pulse of 120. A white count was 21,400 on December 22 with a differential of neutrophils, 46 per cent; lymphocytes, 23 per cent; eosinophils, 1 per cent; monocytes, 30 per cent. She was given on this day an intravenous injection of neoarsphenamine 0.45 gram. This was repeated on December 24. The condition of her mouth and throat was not improving and she began to complain of dysphagia. December 27 there was a sloughing ulcer found on the hard palate and her face was very edematous. She began to be slightly confused. For the past week her temperature had been about 102°F. with gradually increasing pulse and respiration. On December 29 the condition of the mouth was much worse and the teeth were loosening. The white count had risen to 212,000. On the following day blood examination showed red blood cells, 2,200,000; hemoglobin, 50 per cent; leukocytes, 246,600; differential showed neutrophils, 54 per cent; lymphocytes, 13 per cent; monocytes, 11 per cent; myelocytes (?) 22 per cent. On December 31 the rectal temperature was 103°F.; pulse, 120; respirations, 40. Her condition was very poor with much mental confusion. Her face was much more edematous and her spleen was palpable. January 4, 1932, red blood cells, 2,120,000; hemoglobin, 35 per cent; leukocytes, 218,700; differential: neutrophils, 48 per cent; lymphocytes, 32 per cent; myelocytes (?) 20 per cent. The blood culture was negative. January 5 a differential count showed neutrophils, 15 per cent; lymphocytes, 5 per cent; monocytes, 80 per cent, among which there were 2 per cent histiocytes. The monocytes and histiocytes were very characteristic and most of them showed pseudopods. This smear was seen by Dr. Dameshek who felt that there was no doubt as to the diagnosis of acute monocytic leukemia. January 6, temperature, 98.6°F. rectal; pulse, 100; respirations, 40. A differential showed neutrophils, 18 per cent; lymphocytes, 14 per cent; monocytes, 30 per cent; histiocytes, 38 per cent. Platelets were much reduced. She became progressively dyspneic; the sloughing of the palate continued to increase as did

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the edema. On the following day she became comatose and expired.

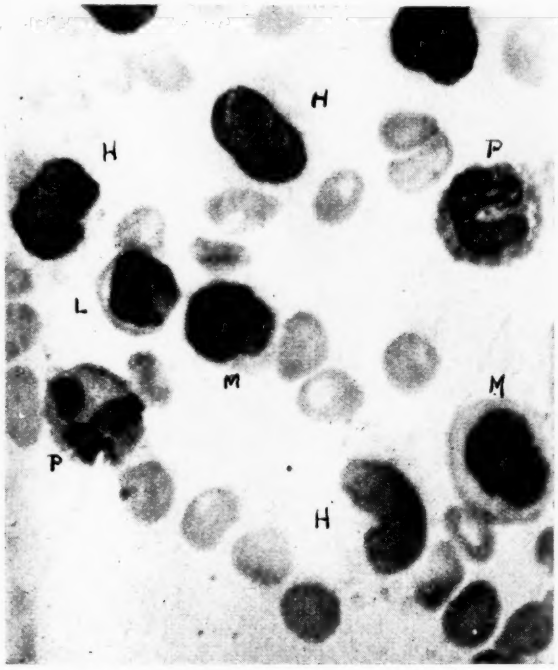
POSTMORTEM EXAMINATION (By Dr. Charles F. Branch, pathologist, Massachusetts Memorial Hospitals and Salem Hospital). The body was that of a well-developed, moderately obese, white, adult female. Rigor mortis complete. Slight postmortem lividity of the entire body. Moderate edema of lower extremities. No signs of recent injuries or wounds. Scalp, ears, and eyes negative. Pupils equal and regular; 6 mms. The mouth was in an advanced stage of gangrenous stomatitis and the right side of the face was markedly swollen. The right ala nasi presented a small, crusted elevation 5 mms. in diameter. Skin over the entire body was marked with minute petechial hemorrhages, each approximately 1 mm. in diameter. Glands of the neck, axilla and inguinal region were firm, hard, enlarged and palpable. In the midline in the abdomen extending from the symphysis pubis upwards for a distance of 10 cm. was an old, healed, surgical incision about which there was a moderate degree of scarring.

Abdominal Cavity. Contained no abnormal fluid or adhesions. Mesenteric lymph nodes were enlarged, firm, light reddish-brown. On sectioning, presented a moist surface. Appendix was absent. Diaphragm on the right was at the third interspace; left fourth.

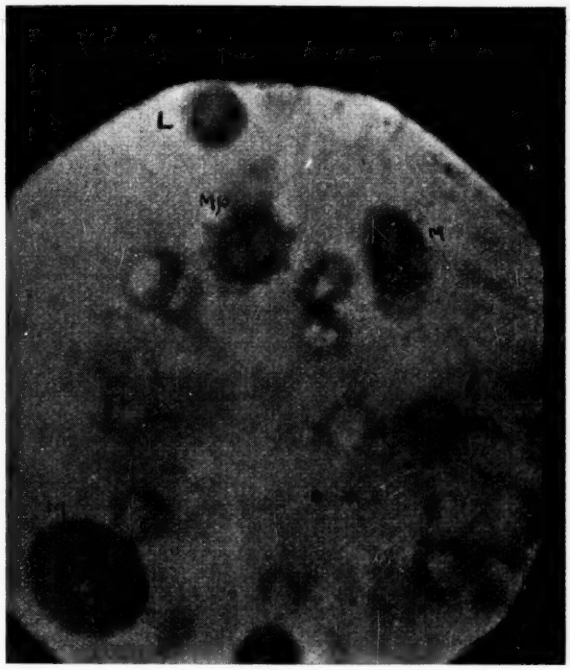
Pleural Cavities. Contained a few, old, fibrous adhesions between the visceral and parietal pleura along the posterior margin.

Pericardial Cavity. Was practically completely obliterated by numerous soft, fibrinous, bright reddish-yellow adhesions between the visceral and parietal pericardium. These were quite firm and showed a moderate degree of organization. In their interstices was a small amount (approximately 40 cc.) of a thin, sanguineous, watery fluid.

Heart. Weighed 500 grams, measured 15 x 12 x 8 cm. Epicardium was completely roughened by partially-organized, fibrinous adhesions above mentioned. Myocardium was rather soft, pliable, light reddish-gray, showed a few irregular mottlings. Just beneath the endocardium were pale, yellowish-gray, fatty deposits. Endocardium was essentially negative. Valves and coronaries negative. Foramen ovale closed. Valve measurements: tricuspid valve, 13.0 cms.; pulmonic valve, 8.0 cms.; mitral valve 11.0 cms.; aortic valve, 7.5 cms.; right ventricular wall, 1.8 cms.; left ventricular wall, 0.3. *Microscopical examination.* Pericardium was covered with a dense network of organizing fibrin, in the meshes of which were large numbers of monocytes, lymphocytes and what appeared to be plasma cells. Connective tissue throughout the heart was infiltrated with many similar cells.



Photomicrograph; oil immersion lens; × 1000.
Histiocytes, H; Monocytes, M; Lymphocyte, L; Polymorphs, P.



Photomicrograph; oil immersion lens; × 1000.
Monocytes, M; Monocyte with pseudopods, Mp; Lymphocyte, L.

Lungs. Right weighed 540 grams, measured 23 x 14 x 4. Pleura was roughened along its posterior surface by old, fibrous adhesions and at irregular areas elsewhere by soft, friable, yellowish-red, fibrinous adhesions. Lung per se was soft, spongy, crepitant, mottled various shades light to dark red. Cut surface showed the alveoli well defined against a light pink to dark red background; slightly moist. Bronchi negative. *Left* weighed 460 grams, measured 22 x 15 x 4. Posterior surface roughened by old, fibrous adhesions. Otherwise, the pleura was smooth and glistening. The lung was soft, spongy, crepitant. Cut surface showed the alveoli well defined against a slightly pink, slate-gray surface which was slightly moist. Bronchi negative. *Microscopical examination.* Pleura was covered with a thin network of fibrin, in the meshes of which were a few neutrophils and many monocytes. The lung per se was negative, although the inter-alveolar capillary spaces were widely distended with monocytes.

Spleen. Weighed 375 grams, measured 16 x 11 x 3.5, was firm, smooth, regular, dark reddish-brown. Cut surface showed germinal centers and trabeculae well defined against a light reddish-brown background. Slight amount of pulp on scraping. *Microscopical examination.* All along beneath the capsule were deposits of fibrin. The entire parenchyma was densely infiltrated with monocytes. In

and about small arterioles had occurred small hemorrhages and much fibrin.

Liver. Weighed 2125 grams, measured 24 x 22 x 8. Firm, smooth, regular, dark chocolate-brown. Normal lobulations well defined beneath a capsule which was not thickened. Cut surface showed normal lobulations sharply accentuated against a dark reddish-brown background. Between the lobules, the interstitial tissue was pearl-gray and stood out prominently. In the lower margin of the right lobe there were a few plaques of bright yellow, soft, necrotic tissue representing minute areas of degeneration. *Microscopical examination.* Section from one portion of the liver showed the region of many of the central veins completely replaced by accumulations of fibrin and red cells. Opposite in this area, sinusoids were so markedly distended with monocytes that the liver cells were rapidly undergoing various stages of degeneration, and infiltration with neutrophils and endothelials.

Gall Bladder. Negative. No stones. All ducts patent.

Gastrointestinal tract. Negative throughout its entire course. Peyer's patches were not enlarged.

Pancreas. Negative in gross. *Microscopical examination.* Massive infiltration of all capillaries and supporting connective tissue by large numbers of monocytes.

Kidneys. Right weighed 250 grams, 15 x 7.5 x 3.5. Was soft, pliable, smooth, regular, light pinkish-gray. Cut surface showed normal relationship between pyramids and cortex poorly defined against an irregularly-mottled, bright pink to slate-gray surface which was moist and bulged slightly at the capsule. Cortex 1.5 cm. wide. Capsule stripped with great ease leaving a smooth, glistening surface. *Left* weighed 275 grams, measured 14 x 7.5 x 4. Similar in every respect to its fellow on the right. *Microscopical examination.* Normal histological architecture was very largely displaced by a massive monocytic infiltration.

Adrenals. Negative in gross. *Microscopical examination.* Normal histological architecture was very largely displaced by a massive monocytic infiltration.

Bladder. Negative in gross.

Genitalia. Removed at previous operation. Nothing remaining but a well-healed, cervical stump.

Aorta. Showed a few scattered atheromatous plaques throughout its entire course. *Microscopical examination.* There was a moderate fibrous and hyaline thickening of the intima, beneath which were deposits of atheromatous material. The adventitia was infiltrated with large numbers of monocytes.

Thyroid. Negative in gross. *Microscopical examination.* Showed remarkably little invasion of monocytes but was otherwise essentially negative.

Buccal mucosa, microscopical examination. Showed the surface completely eroded and covered with a dense network of fibrin, in which were massive accumulations of organisms, the majority of which conformed to those associated with Vincent's angina. The underlying tissues were massively infiltrated with monocytes and a few inflammatory cells.

Femoral bone marrow, microscopical examination. Markedly hyperplastic; showed numerous megakaryocytes, but the outstanding cell was the monocyte.

Mesenteric lymph node, microscopical examination. Showed no remaining normal histological architecture, the whole having been replaced by a massive monocytic infiltration.

Brain and cord, not removed.

Blood throughout the entire body was semifluid, thin and watery. Femoral bone marrow was markedly hyperplastic, dark crimson and filled the entire cavity. Vertebral bone marrow was firm, dark reddish-brown.

Pathological diagnosis. Acute fibrinous pericarditis. Heart hypertrophy. Acute fibrinous pleuritis. Old healed pleuritis. Acute parenchymatous nephritis. Splenic tumor. Femoral bone marrow, marked hyperplasia. Acute monocytic leukemia.

Microscopical diagnosis. Acute fibrinous pericarditis. Acute fibrinous pleuritis. Spleen, petechial hemorrhages. Liver, extensive central necrosis. The normal histology of all of the parenchymatous organs had been materially disturbed by massive monocytic infiltration similar in every respect to any other severe type of leukemia. Blood, acute monocytic leukemia.

SUMMARY AND COMMENT. This patient was apparently suffering from monocytic leukemia of questionable duration. Certainly the process was very rapid, but as to whether it followed the acute respiratory infection or was present at the same time must still remain in doubt. Possibly the signs and symptoms of the respiratory infection were part of the picture of the monocytic leukemia. The first evidence of the blood condition was on December 1, one week after admission, when the differential showed 13 per cent monocytes. The white blood cell count at that time was practically the same as on admission, when the monocyte percentage was only 6 per cent. Clinically her first characteristic symptoms developed on December 19 when the throat condition became evident, although she had enlarged posterior cervical glands on admission. Three days later the monocytes had risen to 30 per cent and by the end of another week the white count had risen to 212,000. The maximum white count was reached on December 30 with 246,600. It is interesting to note the rapid increase in the histiocytes as the disease reached its termination.

Comparing this case with those that Dameshek reviewed, there appear many points of interest. The cases have been shown to occur almost entirely in males although there were two reported in women, this apparently being the third. This case falls within the age group of 5 to 78 with most of the cases occurring between 30 and 50. The fever, rapid course, and gingivitis—those symptoms which point to the disease as an infection—were evident in this case. As in the other cases the blood culture was negative. Previous descriptions of this condition are certainly exemplified by this case with its gradual onset, fatigue and swollen gums, as well as the rapid development of the disease and ulceration of the gums. The fever became sustained, pallor developed, the gums became necrotic and the spleen became palpable with petechial spots appearing just before death. The disease, in this particular case, assuming that it was present at the time of admission, or shortly thereafter, lasted about six weeks. The duration of the disease therefore, was well within that of two weeks to six months as found in previous cases.

As in all other such cases of this condition, the diagnosis was made by the blood examination. There was, as usual, a secondary anemia. Hemo-

globin readings in other cases have been reported as low as 14 per cent and the red count as low as 900,000. The white blood cell count was elevated, although to a lower level than in the other two types of leukemias, most of the counts having been about 20,000 until the late rise to 212,000. Other reports have shown counts ranging usually from 15,000 to 45,000 with occasional high counts, as 400,000. On examining the stained smear the differential count was the interesting feature with the large number of monocytes and histiocytes increasing from 6 per cent on admission to 80 per cent just before death.

The monocyte corresponds in size to the neutrophil or large lymphocyte (10-15 microns). It is oval and at times shows pseudopods. The cytoplasm is grayish blue and contains innumerable, very fine lilac granules. The nucleus is relatively large, usually kidney shaped, has a fine network of chromatin and has no nucleoli. The oxidase reaction shows fine, sparse, positive granules.

The histiocyte, generally accepted as the forerunner of the monocyte, is a much larger, polygonal cell, 15-40 microns, is variable in shape and usually has pseudopods; the cytoplasm is sky blue, contains coarse azure granules grouped about the nucleus and often contains vacuoles and phagocytosed material. The nucleus is about half the size of the cell, is round or oval, contains spongy chromatin and rarely nucleoli. The oxidase reaction is negative. This cell appears in the peripheral blood only when there is an irritation of the reticulo-endothelial system.

For a full discussion of the monocyte, histiocyte and the conception of monocytic leukemia, the reader is referred to two excellent articles by Dameshek[1, 2].

In the differential diagnosis, the progress of the case and the appearance of large numbers of histiocytes exclude benign monocytosis; careful study of the cell type eliminates the possibility of acute leukemia of either of the other two types; granular cells have not completely disappeared as in agranulocytosis.

Postmortem examination confirmed the diagnosis.

Review of Literature. Dameshek[1] reviewed the literature from the original description of monocytic leukemia by Reschad and Schilling-Torgau published in 1913. Any further review of the literature prior to his article would be superfluous. In his paper he carefully analyzes the cases discussing clinical etiology, onset, diagnosis and pathology. He shows in his section on diagnosis what he considers to be the differentiating points between monocytes, large lymphocytes, myeloblasts, premyelocytes and neutrophils.

John S. Lawrence, A. I. Josey, and M. W. Young[3], reported the use of supravital staining along with other well-known procedures for differentiating white blood cells in the study of three patients who had leukemia of the monocytic type.

Cooke[4] reported another case of monocytic leukemia. His patient was a boy, aged 19 years, who had always been healthy until August, 1930, although his family had noticed that he had looked pale for two months previously. When seen on September 2 he had had occasional rises of temperature—up to 100°F.—for the past three weeks had become markedly pale and had some epistaxis and bleeding from the gums. The tonsils were large and the gums swollen and granular. The spleen was enlarged. The superficial glands were not enlarged, nor were there any petechial hemorrhages. Blood examination on that date showed red blood cells, 1,260,000; white blood cells, 49,800; hemoglobin, 22 per cent; differential: neutrophils, 4 per cent; lymphocytes, 9 per cent; monocytes, 87 per cent. Thrombocytes were scanty. The patient died on September 12. Postmortem examination was not allowed.

The present case has been reported not because it shows any features not already fully discussed by Dameshek but because it adds another case of monocytic leukemia to the twenty-two previously reported. It is interesting from the fact that it appeared in a woman, ran a very acute course and came to autopsy, adding a complete pathological report to the previous eleven which have been obtained.

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CANADA AHEAD OF US

A recent bulletin from the Canadian Tuberculosis Association makes a very interesting statement, viz., that Canada has reached, and even surpassed, the standard of one bed to each annual death. The figure for Canada is 1.05 beds per death. This is a very remarkable achievement and deserves high praise for the earnest and successful efforts of the Canadian Tuberculosis Association along these lines. In contrast to this figure, the ratio of beds to deaths in the United States has reached only 0.91 beds per death. The United States still needs 8,000 beds to reach the minimum standard already attained by Canada.—*Bulletin National Tuberculosis Association*, October, 1932.

NEW HAMPSHIRE MEDICAL SOCIETY

THE ATTENUATION OF MEASLES*

BY COLIN C. STEWART, M.D.,† HANOVER

MEASLES is so universally accepted as an inevitable visitation, and is commonly so mild, that parents are inclined to disregard its possible serious consequences. Thirty years ago, when scarlet fever was a cause of grave anxiety, an attack of measles was often regarded as a subject for mirth. Today, due to the inexplicable decrease in virulence of scarlet fever, measles is the more serious menace to childhood. In London there are now six deaths from measles to every one from scarlet fever[18]. Herrman[15] found two and four-tenths per cent of all deaths below the age of ten, and six per cent between one and three, due to measles.

Ninety per cent of the deaths occur under five years of age[29], so that many lives would be saved if the illness could merely be postponed. Yet under certain conditions measles can also be a menace to adults, as illustrated by the epidemics in training camps during the War. Since 96-98 per cent of susceptible contacts develop the disease after intimate exposure[34], it is rarely possible to prevent spread to other children in a family by isolation alone.

In recent years a number of investigators have been seeking a means of reducing the mortality of measles. As early as 1896 injection of convalescent serum during the course of the disease was tried and found to be unavailing[33]. Weaver and Crooks[32] have demonstrated again that even large doses given after the onset of the symptoms are without appreciable effect. However, when injected during the incubation period convalescent serum has a pronounced effect. Nicolle and Conseil[24] discovered this in 1916, and were able to completely protect a child from measles. Their report is generally cited as the first published account of the prophylactic use of convalescent serum. Siegel and Ermann[29], however, say Cenci used it as early as 1907. In recent years many reports have appeared which have been uniformly favorable[19, 25, 28]. Convalescent serum is today the most effective agent in controlling measles, and if given early, in adequate dosage, the disease can usually be prevented. The number absolutely protected is variously given as from 84 per cent to 98 per cent[4, 13, 21, 23, 25, 31]. Most of the remainder developed only a greatly modified form.

The usefulness of convalescent serum is unfortunately restricted because of the difficulty in obtaining an adequate supply. Parents not unreasonably object to having blood drawn from their convalescent children, except perhaps to help a younger brother or sister. Adult measles convalescents are seldom available outside the larger cities, and, even in New York, Park and Freeman[25] were unable to obtain the amount of serum they needed.

There is thus a need for a more practical method of prophylaxis. As a preliminary step, the causative agent of measles has been sought by a host of workers, and several diverse organisms have been isolated from patients. Thus Tunnicliff[30] and Caronia[6] have found dissimilar anaerobic diplococci; and Ferry and Fisher[12] have isolated an aerobic streptococcus which they have named streptococcus morbilli. Sera have been prepared with all of these various organisms. In addition Degkwitz[10] has an anti-measles sheep serum made from a filtrate of the nasal secretion and sputum of measles patients. Even normal horse serum has been found to give partial protection[20]. All these workers have reported favorable results from the prophylactic use of their preparations, but reports by others are conflicting. Most observers have found convalescent serum superior to any of the animal sera available, not only in respect to the number of patients protected, but also because of the absence of serum sickness. This work gives promise of producing a preparation which will be as effective as and more readily available than convalescent serum, but final judgment must await further studies.

In 1921 Reitschel[27] advocated the use of serum or whole blood of adults who had had measles, when convalescent serum was not available. Degkwitz[9] had previously tried this, but gave it up because he thought the immune bodies were not present in the blood in sufficient concentration. The favorable reports which followed Reitschel's paper have been well summarized by Morales and Mandry[21]. These authors find that results comparable to those with convalescent serum are obtained if 20 cc. or more of immune adult serum is used. Zingher[34] found it necessary to use about three times as much serum from an adult who had had the disease several years previously as from a convalescent. The unfavorable results of Blauner and Goldstein[5] are absolutely at variance with the experience of others. This is possibly due to a lack of even approximate knowledge of the dates of exposure of their patients, with a consequent inability to give the injections at the most favorable time.

The donor of course should be in good health. One of the parents is usually most available, but when a choice is possible, older children in

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†Stewart—For record and address of author see "This Week's Issue," page 801.

the same family, who have had measles more recently, should be preferred; and adults who have had measles within ten or fifteen years should be better than those long over the disease[3]. There is some evidence that serum from people constantly in contact with measles, as doctors and nurses[3, 17] is more effective. Attempts have been made to reactivate the blood of adults by vaccination before using it to protect children, but results have not been very encouraging[1]. It seems reasonable to expect some dilution of the antibodies in the blood of a professional donor, possibly another reason for the unfavorable results of Blauner and Goldstein[5].

Debré and Ravina[7] in 1923 emphasized that because of the short duration of passive immunity, active immunization by a mild attack of measles should ordinarily be preferred to complete protection. Immunity is then lasting, whereas the effect of an injection which results in the production of complete temporary protection lasts only about two months[34]. Active immunization has also been attempted by repeated small injections of infected blood. Hiraishi and Okamato[14] observed no benefit in their experiments, but Debré[8] and his associates have reported encouraging results with a slightly different technic. Degkwitz[11] injected cultures of conjunctival and pharyngeal secretions in an attempt to produce active immunization. Herrman[15] succeeded in immunizing children by transferring infected material from the nasal mucosa of early measles patients to the noses of babies four and five months old, when there is still a natural immunity. This procedure has not become popular because of the possibility of transferring a virulent measles, as well as the chance that other infections might be given the baby[19].

The method of active immunization described by Debré and Ravina[7] has been most widely followed. They advocated the modification of the measles attack through injections of convalescent serum late in the incubation period. It has been shown since that the blood of adults who have had measles will answer the same purpose, if used in large enough amounts. Since less is needed than to prevent the attack, the large size of the injection is not so much of an objection; and the availability of such blood makes it particularly useful for this purpose.

Measles as modified in this manner is characterized by a long incubation period, usually from 12 to 21 days but occasionally even longer. Catarrhal symptoms are absent and Koplik's spots are seldom found. There is occasionally a mild prodromal rash. Fever is slight, and the eruption is faint and of short duration. As the eruption fades, it may become ecchymotic[26]. The enanthem occasionally simulates follicular tonsillitis, with a fever to 103° or even higher; but this lasts only about 24 hours[16]. Complications are infrequent.

We have, then, a means at our disposal of lessening the mortality from measles. Yet it is not widely used. This is probably because of the difficulty of obtaining convalescent serum, coupled with a failure to appreciate the value of the blood which can be readily obtained[2]. The choice of method may well vary with the circumstances. In an institutional epidemic, the necessity of stamping out the disease outweighs the advantage to the child of an attenuated attack. For this purpose, convalescent serum should be used if it is available, because the rather large amounts needed for absolute protection favor the most concentrated product. If adult blood is to be used, it is worth while to reduce bulk by separating the serum, or by using plasma as advocated by Park and Freeman[25]. The technic of preparation has been discussed by several authors[16, 22, 24]. It is possible to store the serum or plasma for as long as three months, but after that time it is well to secure a fresh supply.

For use in private homes to protect the brothers and sisters of a measles patient, the simplest method is the best. It is impossible to wait several days for a Wassermann to be reported, so reliance must be placed on a negative history. Blood is withdrawn from the arm of the donor with a 20 cc. Luer syringe, a sterile needle is attached, and the blood is immediately injected into the gluteal muscles of the child. The use of a large needle facilitates the operation considerably. Two cc. of a five per cent sodium citrate solution drawn into the syringe before taking the blood eliminates the necessity of hurrying.

The amount of blood to be injected depends upon the interval since exposure, the age of the patient, and the result desired[16]. Apparently convalescent blood is effective in amounts approximately one third as large as the amount which must be used if the donor has not recently had the disease[34]. If serum is used, the dose can be cut in half. For older children, the amount should be somewhat larger. Twenty to 40 cc. of parental blood is the accepted dosage for attenuation, in a small child. For complete protection, at least twice this amount is needed.

It is best to inject during the first five days of exposure for complete protection. However, with an increase in the dosage, injections as late as the eighth or ninth day may be effective[16, 34]. For attenuation, it is usual to delay until the sixth to the ninth day; but it is logical to suppose that, if given earlier, the same thing could be accomplished by a smaller dose.

I recently had occasion to test the prophylactic effect of whole parental blood. Three adjoining families, having among them nine chil-

dren aged from two to eight years, were quarantined with pertussis. The older children had their lessons in the three houses by turns; and the entire group played together. One morning one of the children woke up with a typical measles rash. If we assume the period of invasion to be four days, the eight other children were then in the fifth day of the incubation period.

Because three of the children were quite small, and because all were convalescing from pertussis, it was thought best to give injections of parental blood. That same day, two brothers of the patient were given intramuscular injections of their mother's blood. Another brother was said to have had measles and was not treated. The following day the three children of the second family were similarly treated; and next day, the seventh day of exposure, the last two children had their injections. Twenty cc. were given except to the last child, who had only 10 cc.

On the fourteenth day of exposure the one child who had escaped a prophylactic injection developed a typical measles rash, which lasted five days. Like the first case, this was an ordinary mild measles. On the evening of the fifteenth day he had a rash which lasted only 24 hours, without fever or other symptoms of illness. The next day the fourth brother also had an eruption, which had practically disappeared next day. This child was the only one of the entire group given blood, who felt ill at any time. He had slight fever for two days during the period of invasion; and his tonsils were swollen and inflamed. No Koplik's spots were seen. On the seventeenth day of incubation, the three children of the second family had eruptions. One was quite transitory, and only on the face; one lasted a day and a half; and the third, which later became ecchymotic as described by Regan[26], lasted four days. None of the children had any symptoms. The last two children, injected on the seventh day, were reported to have slight rashes on the nineteenth day only. They were also symptom-free.

It is curious that the incubation period was prolonged most in the children having their prophylactic injections latest. With so few cases, no definite conclusions can be drawn from this observation.

CONCLUSIONS

Under conditions of private practice it is well worth while to protect the brothers and sisters of a child with measles by intramuscular injections of blood from some older member of the family who has had measles. This is especially true of the younger children. Unless a child is already seriously ill, it is wisest to attempt to produce an attenuated form of the disease, which presumably gives lasting immunity[33]. Twenty cc. of whole blood can easily be injected without delay or complicated equipment. The history that a child has had measles should not be too thoroughly relied upon.

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DISCUSSION

DR. PAUL DYE, Wolfeboro: I do not wish to discuss the paper, but I would like to ask a question of Dr. Stewart. Does the injection of serum give active immunity?

DR. STEWART: It depends on how it is used. Injected early, in sufficient quantities, it entirely prevents development of the disease, by con-

ferring a temporary passive immunity. If a small amount is given, however, the usual result is an attenuated form of measles, which produces active immunity.

DR. ABBOTT L. WINOGRAD, Nashua: I would like to ask if there is any way one can be sure that passive immunity can be brought on. Of course, if you give an injection of whole blood sometimes you find you get quite a clot in the buttocks that refuses to yield for a month or so and may sometimes break down and become infected; and if you do not get active immunity I wonder whether sometimes it is worth while trying to prevent development of the disease except in the youngest children, for example, where pneumonia may follow, as in children a year old.

Another question: Is commercial measles serum effective? One more point, if I am not presuming too much, is whether the doctor has had some experience with transference of infective nasal secretions to the mucous membrane of a child who has not been affected, and whether that seems a practical, although easy, method?

DR. STEWART: Injections of whole blood, either in this or other conditions, are seldom followed by a reaction of any consequence. Infections are practically non-existent, unless sterilization of instruments has been inadequate. As to the question of a clot forming, whole blood injected into the buttocks is ordinarily rapidly absorbed.

During the first years measles frequently results in broncho-pneumonia, which accounts for most of the deaths. The mortality is less after infancy, but is still considerable up to five or six years of age. It certainly seems that we should do all in our power to protect children of this age group against measles, even if some will acquire only a temporary immunity.

Commercial sera and transference of nasal secretions were discussed in the early part of the paper. Serums have been prepared with various organisms. Some have been reported on favorably by the originators, and by some of their associates. Other investigators have had less success with the same preparations. In no case do they seem to give as good results as the convalescent serum, although one investigator has reported a very high percentage of prevention in a recent article. Immunizing babies by the transference of nasal secretions has not become popular, because of the danger of producing a case of virulent measles, although Herrman reports very good results.

SOME PATHOLOGICAL PROBLEMS*

BY ALBERT C. JOHNSTON, M.D.†

CASE I

THIS was a white female, aged 33, mother of five children, who had been previously admitted for pains in the tibia, irregular temperature and secondary anemia of unknown origin. A diagnosis of floating kidney was made at that time.

The patient had left the hospital much improved, no more pains in the tibia, appetite was better and her blood picture was improved. She went home with liver extract as medicine. When she left she still had occasional high fever, and her supposedly large floating kidney on the right side was still very apparent. At home she felt well for two or three weeks, was walking and visiting. A month after she had left the hospital, I was notified that they were rushing her back in an ambulance and in a very serious condition. When she arrived in the evening, she had considerably changed since I had last seen her.

Examination: Her facies was that of a septic condition. She was very thin and emaciated and weak, with thready and rapid pulse, marked dyspnea, and severe pain all over the abdomen, especially in the right hypochondriac region. In the right upper quadrant a tumor the size of an orange, very sensitive, and movable with respiration, was located at about three fingerbreadths from the costal border; this tumor was apparently intraperitoneal. A consultation was immediately asked for and obtained. The patient being in such a desperate condition, we decided to make an exploratory laparotomy, expecting a large purulent gall-bladder or a large kidney.

Abdomen: Tumor was found in the right upper quadrant.

Genito-Urinary: Negative except probably large right floating kidney.

Neuromuscular: Very emaciated, marked atrophy of arms and legs.

Skin: Brown pigmentation.

Special Examination: Urinalysis, blood count.

Urine had specific gravity of 1010, no sugar, albumin. Microscopical examination revealed fine and coarse granular, hyaline and waxy casts.

Laboratory notes: Hemoglobin 50%, W.B.C. 9200, Neutrophils 80%, Lymphocytes 15%, Endotheliocytes 5%.

Tentative Diagnosis: Large gall-bladder or kidney.

At operation, a tumor of the liver was noted and the patient was closed up.

PROGRESS NOTES

2-12 Patient operated upon.
2-13 Condition better than expected. She seems quite relieved. Believes tumor is removed and is grateful.
2-15 Condition same.
2-17 Condition worse; patient weaker, pulse rapid, wound well healed.
2-20 Family notified of her serious condition. Very cachectic; has constant aggravating cough every time she talks.
2-22 Patient growing weaker, coughs constantly. Narcotics have to be used regularly. Sleepless.
2-27 At 7 P. M. patient was in coma. Died next morning at 2 o'clock.

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AUTOPSY

Two hours after death, the body was opened through the old incision which had healed perfectly. The incision was carried from pubic bone to clavicle through the sternocostal cartilage.

The first thing noticed was a tumor on the liver, a very large and necrotic growth the size of an orange on the diaphragmatic surface of the right lobe.

The spleen was small.

Pancreas, small and not removed.

Right kidney, large. Left kidney very large. Small gall-bladder.

Metastasis to great omentum and mesentery, the same was riddled with nodes or nodules the size of a bean.

On the small intestines near the ileocecal valve, there were signs of beginning gangrene caused by a nodal tumor obstructing the circulation.

Large intestines were adherent and on the sigmoid there was a very small growth similar to liver growth developing in the wall of the sigmoid giving the appearance of a stricture.

Pelvis: Ovaries were large and covered with metastasis of the growth, the tube was also involved. The uterus was normal.

The right chest was full of greenish syrupy fluid, about 500 cc. The lung was collapsed to the apex, no normal lung distinguishable.

Left chest contained a small amount of fluid, the lung tissue was mottled with whitish spots.

Heart: The pericardium has fluid, the right ventricle was very large, flabby, and degenerated.

Liver: The weight was about normal. A nodule in the right lobe of the liver was 9 cm. in diameter, with resistance to cutting, and whitish in color and the consistence of India rubber. There was also a small single tumor, 5 cm. in diameter.

The right kidney weighed about 300 gm. The cortex, pelvis and medulla were filled with whitish tumor.

The ovary was infiltrated with whitish tumors.

MICROSCOPIC EXAMINATION

A celloidin kidney section.

There were many dark stained vesicular nucleated cells in the interstitial tissue and parenchyma of the kidney. These nuclei were for the most part spherical, irregular in size and the chromatin was granulated. A few cells per low power field showed mitotic figures. Cytoplasm of these cells stained pinkish and the cell as a whole was round. Extracellular substance was very slight in amount.

A section from the large tumor of the liver:

No normal liver elements were present. L. P. showed many cells closely packed chaotically with deep blue staining nuclei and, in places, pink. There were areas with very few cells and much C.T. with some nuclei showing karyolysis and karyorrhexis.

Diagnosis: Round cell sarcoma.

This case is of interest because of its rarity and the confusion that exists in the selection of a name for it. A section was diagnosed as lymphosarcoma by a pathologist in Quebec and lymphoblastoma by the surgical pathologist of the Boston City Hospital. As you notice I call it a round cell sarcoma. Personally I think the term "Round Cell" is objectionable as it does not throw much light on the origin and connotes an attempt to cover ignorance. However, a lymphosarcoma is derived from lymph tissue, spreads by contiguity and not by blood stream metastasis, except rarely, and since this evidently metastasized by the blood stream as evidenced by isolated metastases in the liver and lung, the term lympho-sar-coma does not distinguish it from a contiguously growing tumor. Lymphoblastoma includes a great host of tumors of lymphatic origin, benign as well as malignant, including Hodgkin's disease, and to my mind is too general to convey a meaning of the nature of this tumor. Lymphoblastoma is a good term if qualified by proper adjectives because these tumors are truly derived from lymphoid tissue with embryonic characteristics. The point I wish to bring out is that all three diagnoses, though different, mean the same thing when interpreted by each of the diagnosticians concerned. In making a pathological diagnosis however, there is more than the academic position to consider. The surgeon wants a decisive answer to the following questions:

1. Is it malignant or benign?
2. If malignant, how does it spread?
3. What is the prognosis?

Evidently the Boston diagnosis answers none of the questions. Lymphosarcoma is erroneous. Probably blood metastasizing malignant lymphoblastoma is best.

CASE II

This patient, an adult, white, male, 51 years old, was taken ill three weeks ago with sharp pains in the abdomen, vomiting and no bowel movement for four days. He complained of pains in the lumbar region and was unable to sleep since taken ill. He seemed to have had intestinal grippe, but he also had indulged much in liquor. When admitted, he was vomiting fecal matter and looked very ill. He was in the hospital seventeen days and ran a subnormal temperature from 95.4 to 98.2. His pulse ranged from as low as 48 on the fourth day to 120 on the day before death. His respirations remained between 20 and 24 except on the third day in the hospital when they were 42.

PROGRESS NOTES

8-30 The patient did not retain anything. Turpentine stupes were applied to the abdomen. Stomach lavage, Ringer's solution intravenously and Croton oil were employed.

8-31 H.S.S. enema was given and pituitrin repeated. There was a fair result. Nutritive enema was given.

9-1 The temperature continued subnormal. The condition was very poor.

9-4 There was not much more to say except that we gave him nutritive enemas, various stimulants and intravenous injections of nutrient solutions, glucose, buffer salt and Ringer's.

9-6 Same measures continued, condition remained unchanged. There was much difficulty in moving the bowels. He seemed to retain food a little longer but vomited as before. He had to be given opiates once in a while to quiet him.

9-12 The patient's pulse was very weak, the hands very cold and clammy, with profuse perspiration.

9-14 The patient died.

Clinical Diagnosis: Intestinal obstruction due to adhesion.

The autopsy was performed one hour after death confined to the abdomen.

Findings: The large bowel was dilated, measuring 6 cm. in diameter, collapsed about two feet above the ileocecal valve. Moderate fibrinous exudate covered the entire bowel. Intussusception of the bowel was found extending from above downward toward the ileocecal valve. The invaginated portion was about 15 cm. in length. The tumor was sausage shaped about 15 cm. long and 8 cm. in diameter, and sections showed it to be dark red much like a blood clot. The intussusception did not come nearer than two feet from the ileocecal valve.

DISCUSSION

This case is of interest because it occurred at an age at which intussusception is not usually found, the duration of life after the intussusception originated and the curious fact that the attending physician claims to have obtained a very copious movement of the bowels the day before death and seventeen days after the beginning of the obstruction in spite of the fact that postmortem findings showed a complete obstruction and an area of dead bowel. Furthermore I could find nothing to explain the fact that the large bowel was distended very markedly although the obstruction was above it. The only explanation seems to be that there were adhesions of very recent origin at the splenic flexure and at the cecum, which made a closed tube of the cecum, ascending and transverse colon. Besides these findings, and of course the peritonitis, no other gross pathological findings were observed in the abdominal cavity.

CASE III

This is a case of a male, white, aged 48, with the following history.

Present Condition:

Four months ago, the patient called on me complaining of dyspnea and cough. On examination of the chest there were found symptoms of passive congestion, râles and dullness in the bases. The abdominal cavity distended by fluid with marked edema of the lower part of the body, legs and feet. His appetite was good, but he felt weak and had lost weight in the last few weeks. A large, regular smooth prominent tumor was found at the right lower costal border extending as far as the umbilicus. It was thought to be an enlarged or cirrhotic liver. He was sent home and put to bed on a limited diet. The fluid and edema disappeared. The condition not improving, he was advised to enter the hospital for treatment.

On admission, four months after the onset of the disease, he had slight fluid in the abdomen, no edema, good appetite, moderate loss of weight, and was less emaciated. The blood picture showed secondary anemia, the Wassermann test was negative, the urine test was of no significance. Several consultations were asked for and obtained. On physical examination an irregular hard and soft tumor was localized in the right hypogastric region extending to the umbilicus on the right side, movable, painless and moving to and fro with respiration. The clinical picture on palpation was unlike that which was found before.

Provisional diagnosis: Possible malignancy of the liver, cirrhosis, or syphilis.

He was operated on the week following entrance to the hospital.

The temperature was normal except for slight rises a few days after the exploratory operation. The pulse was fairly constant between 60 and 80 until the day after operation when it rose to 140. It was 120 at the end. Respirations were relatively normal. On the ninth day following admission an exploratory operation was performed with a preoperative diagnosis of malignancy of the liver or syphilis.

Detail of Operation:

A right, high rectus incision was made. No fluid was found in the abdominal cavity. A large irregular, spherical tumor was located retroperitoneally extending from the right region of the kidney filling the right hypogastric region and extending to the median line. The tumor was apparently three times the size of a grapefruit. Marked vascularization was noticed over the tumor. The transverse and ascending colon were firmly adherent to the tumor. The left kidney could not be localized. The spleen was found to be indurated and enlarged. The gallbladder was indurated and nodules were found on the liver and they were interpreted as metastases from the kidney tumor. The condition of the patient did not warrant removal of a portion for biopsy. The abdomen was closed.

After the operation the patient became weak and lost blood from the incision for ten days. Two days after the operation, he became much weaker and had severe diarrhea; the pulse was thready and he coughed constantly. During the next six days he became increasingly weaker, diarrhea increased, respirations became more difficult and loss of weight was very marked. He died exactly ten days after the operation.

Urinalysis on the first day of admission showed a slight trace of albumin with blood, leucocytes, fine and coarse granular casts, hyaline and waxy casts. Apparently no attention was attached to these significant urinary findings in establishing a diagnosis. Six days before death the urine was normal. The Wassermann and Kahn tests were negative. The white count was 9,200 with 80% neutrophils, 15% lymphocytes and 5% endotheliocytes.

An autopsy performed an hour after death revealed the following:

A male, about fifty years old. The left pupil was larger than the right. The mucous membrane was pale. A right rectus incision, twelve and a half centimeters in length (12½) was found. There were blood clots in the wound, with blood oozing from the lower third. Filmy adhesions between the peritoneum and surface of the liver and also between the liver and the transverse mesentery were present. There was a large hematoma in the abdominal wall approximately six centimeters in diameter.

The exposed surface of the liver shows smoky infiltration due to fibrous tissues with tumor nodules of pepper grain appearance. The color of the tumor was that of old rose. The growth in the liver constituted one-fifth of the liver substance. The liver extended six centimeters below the costal margin on the right side. The gall-gladder was small. There were broad adhesions between the sigmoid and the peritoneum. The bowels and mesentery appeared normal externally. The cecum was bound down by adhesions, there were adhesions about the appendix, and the posterior wall of the cecum was bound by tough old adhesions. The appendix was bound down to the tumor mass. The transverse mesocolon was adherent to the tumor.

The whole right upper and lower quadrant was filled with a large nodular cystic and hard retroperitoneal tumor of the kidney extending from the crest of the ilium to the diaphragm and from the lateral side to about two cm. to the left of the midline. Closer inspection revealed a tumor covered with large blood vessels. The lateral side was very ad-

herent to the peritoneum. The right kidney weighed four and a half pounds or two kilograms.

The liver cut with resistance and there was a diffuse pink growth disseminated throughout this organ. A section was taken for diagnosis.

No metastasis to bone was found.

A section of the large tumorous kidney revealed that the cortex and the medulla were replaced to about 9/10 by a tumor mass which was pinkish, whitish, speckled with black in parts and yellow. The tumor bulged above. There was one tumorous mass about three inches in diameter which was jet black.

The spleen was slate colored, and about normal in weight. The surface made by cutting showed normal pulp. The spleen was normal.

The left kidney was large and lobulated, of the fetal type. It weighed about 250 grams. The surface made by cutting showed distinct liver marking. The cortex averaged 11 mm. in diameter.

The pelvic fat was normal in amount. There was no hyperemia of the left kidney.

The descending colon was small.

The lung showed hypostatic congestion.

The heart was normal.

The anatomic diagnosis was melanotic sarcoma.

The kidney section showed very little kidney parenchyma. It was replaced by a growth exceedingly rich in large irregular engorged blood spaces. Free blood was also found in the stroma. The tumor was composed of large pale cells with irregular deep-stained nuclei. The arrangement was very chaotic. There were many deposits of melanin in the normal surrounding tissues as well as in the tumor. The tumor cells varied greatly in size and shape, but for the most part were approximately oval. In some places there were many blood spaces with island nests of large cells with unstained cytoplasm and irregular dark blue nuclei. A few glomeruli were seen. There were one or two small areas of lymphocytic infiltration. Mitotic figures were seen.

Section of liver showed the tumor as aforementioned. No normal tissues were distinguishable.

In some sections of the tumor, cells were large, clear with oval nuclei and in strands held together by a connective tissue reticulum resembling very much the structure of hypernephroma. A few large nerve cells were also seen.

Final Diagnosis: Hypernephroma with melanin deposits.

Discussion

Dr. Osmon H. Hubbard, Keene: Yesterday afternoon you had the opportunity of listening to a paper by Dr. Metcalf on Post-Mortem Lethargy, with a discussion by Dr. Miller and Dr. Parker, and this morning we have an excellent example of what Dr. Miller brought out in regard to the value of autopsies. Dr. Johnston is to be congratulated, I think, on being able to do autopsies on these three rare, unusual and interesting cases and completing their records by his very excellent reports.

A great many cases occur, and you all meet with them every little while, where the diagnosis is in doubt, or where it is impossible to make a diagnosis by the clinical symptoms, as in these cases which have been reported, and it is a great satisfaction, not only to the physician, but to the relatives and friends to know the exact conditions which caused death, and to appreciate perhaps that everything possible had been done for the patient, and that no more relief could have been afforded. It also adds to the store of knowledge of the physician from which he can draw in dealing with future cases and adds greatly to the accumulated value of medical knowledge to have these cases completed by autopsy.

At the time I was Medical Referee I had many cases of sudden deaths. They were deaths from natural causes, apparently, but the cause of death was rather indefinite, and, while there was no reason for the County Solicitor to order an autopsy, I made it a point to try to obtain one in these cases so as to decide definitely the cause of death rather than give it in a general way, perhaps, as heart disease, or cerebral hemorrhage, or something of that nature, because I did not know the exact cause. We had a number of interesting cases, and I had no trouble in obtaining orders for autopsies from him or getting permission from relatives. In fact, in one or two cases I was requested to do an autopsy by the relatives in order to decide the immediate cause of death.

In one case the cause of death would have been recorded as simply heart disease from a knowledge of the case and its history, but the autopsy showed it to be rupture of the heart through an old infarct, the rupture being large enough through which to put a lead pencil.

In another case a man was found dead in bed, whether from cerebral hemorrhage or some other cause was not known. The autopsy showed an occluded coronary artery.

Still another case was that of a man who had been away visiting. He evidently had been having a very good time, and when he reached Keene the conductor thought he was intoxicated and had him removed to the police station. He was a young man, only 29 or 30 years old, a very rugged, strong person. The next morning, instead of recovering, he was in an even worse condition and after a few hours died. I was able to do an autopsy a few hours later, and examination of the body showed no pathology in the abdomen or in the chest, but on opening the skull the brain presented a congested condition with a little bulging over the right ventricle and a few small clots; on making a small incision the blood poured out from the ventricle, showing a cerebral hemorrhage into the right ventricle from some of the veins in that region, which had caused his death.

I think we should try to obtain more autopsies even if they are not so puzzling as those which Dr. Johnston has cited to us, and they would clear up a great many of these cases which are more or less obscure.

Dr. R. E. Miller, Hanover: I would like to express my appreciation for Dr. Johnston's careful pathological work and for his clear presenta-

tion of it. I am interested particularly in Case I, which apparently belongs to that well-defined group of lymphoblastomas described in Dr. Marshall's classification of that group of tumors, a group which pathologists and clinical men have always had some difficulty in classifying. The group is fairly well defined. An accurate and proper name for the group has not yet been found, as Dr. Johnston pointed out.

I would like to use this opportunity to make it known to you that Dr. Callender is in charge of a committee of the Association of Pathologists and Bacteriologists which has as its office the classification of tumors of this growth and of cases of lymphatic leukemia, that is, cases that come into the lymphatic group. This committee is trying to have all such cases which have been carefully worked up and had postmortem examinations reported to it, with the idea of adopting a uniform classification and nomenclature. This case is one that certainly would fit into the requirements made by Dr. Callender and the committee.

Dr. George S. Foster, Manchester: *Mr. President*—It seems to me we have been most fortunate this morning in listening to Dr. Johnston on this subject, and I feel that there is no man in any line of medicine who should not be interested enough to learn some pathology. The importance of postmortems I think cannot be overemphasized, as covered by Dr. Metcalf yesterday. I believe that with postmortems we can more readily appreciate our errors in diagnosis. It brings them to us and this is a most important matter when dealing frankly with our mistakes.

I feel also that while we are doing postmortems we should, of course, secure specimens of the tissues so that from these specimens, sections may be made for study. I hope sometime, looking not too far into the future, that our own state will have in each community some man who is a student of pathology. Thus can be rendered a service of inestimable value. It is also hoped that we will have equipment in each community where pathological section work can be done. Thus following postmortem examinations we can submit these specimens, and have the sections made and at least secure a tentative diagnosis very early.

I also look farther into the future and hope some time to find that this very group will take up this line of study. I believe it could come from the clinical study of medicine, which would interest a group large enough so that they could meet perhaps once or twice a year and discuss this subject. This is a very important matter if we are going to reach the goal of definite diagnosis. I think we are to be congratulated in having the opportunity of listening to Dr. Johnston's paper.

Dr. Johnston: I have been very much aided by Dr. Miller and have learned quite a bit from him. I was wondering why some such committee had not been formed by the Association of Pathologists for a definite stand on diagnosis. It is rather confusing to a doctor who wants a report on a specimen, sends it to a first-class pathologist and gets one name, to get a different designation when he sends it to another. He does not know what to believe. Of course, these names should convey to the surgeon who wants the information, some idea of the degree of malignancy, because many times these sections are sent with the expectation of future benefit and not merely checking on whether the diagnosis should be lymphoblastoma or round cell sarcoma.

MISCELLANY

HILLSBOROUGH COUNTY MEDICAL SOCIETY

At the Annual Meeting of the Hillsborough County Medical Society, held in Manchester, October 25, Dr. Francis M. Rackemann of Boston gave the principal address.

Officers elected for the ensuing year are Dr. H. M. Morse of Peterborough, President; Dr. T. F. Rock of Nashua, Vice President; Dr. D. G. Smith of Nashua, Secretary-Treasurer. Dr. E. A. Jones of Manchester was elected a member of the board of censors; delegates to the State Society, Dr. George C. Wilkins of Manchester and Dr. D. G. Smith of Nashua for two years. Dr. D. W. Parker of Manchester was reëlected to the advisory committee on jurisprudence.

NEW MEMBER

Dr. Frederick S. Gray, 350 State Street, Portsmouth, N. H., graduate of Columbia University 1929, licensed 1932.

MEDICAL PROGRESS

PROGRESS IN THE SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM IN 1931*

BY JAMES C. WHITE, M.D.†

IN preparing this year's review of progress in sympathetic neurology, the writer has gone over some one hundred and fifty papers and selected forty-eight for review, as these have the most direct and important bearing on the problems of medicine and surgery. During the past decade in which a renewed interest in the involuntary nervous system has been active, operative intervention has already been tried on nearly every condition which could conceivably be controlled by the protean activities of these nerves. As a result there have been no outstandingly new procedures reported in the past year, but there has been much interesting work in physiology and a more critical sifting of previous surgical procedures.

I. Physiology

Some extremely interesting studies have been made by Heymans and his collaborators[1] in Belgium on reflex regulation of the general circulation by the carotid sinus. They point out that there is a very special innervation of the region in which the common carotid divides into its internal and external branches; from this bifurcation are given off many autonomic nerves which go both to the vagus and the cervical sympathetic trunk. The following summary of their work has appeared during the past year:

1. Carotid sinus regulation of blood pressure. At normal arterial pressure, variations of tension of only 10 to 20 mm. of water in the carotid sinus produce marked fluctuations in general systemic blood pressure. When the carotid sinus pressure is raised, the systemic pressure falls. At pressures above 200 mm. and below 50 mm. of mercury, reflex control of the vasomotor tone disappears. The authors have demonstrated that between these limits a rise in carotid pressure will produce peripheral vasodilatation and a fall in general systemic blood pressure. There is also an increase in the size of the spleen and of the intestines along with a diminution in rate of blood flow and an increase in cardiac volume.

2. Reflex regulation of heart and lungs by the carotid sinus. These authors have also shown by a long series of experiments on dogs that the carotid sinus has an important reflex control on the activity of the heart and lungs. Perfusing the carotid sinus with blood containing varying concentrations of carbon dioxide and other weak acids causes characteristic acceleration of respiration, even though the blood flowing through the brain is unaltered. In the case of the heart also, marked changes in its rate and the force of its beat can be produced by perfusing the isolated carotid sinus with adrenalin.

Bronk[2] has further elaborated on Heyman's studies. By using a vacuum tube amplifier and a string galvanometer attached to the carotid sinus plexus, he has been able to show a burst of nerve impulses accompanying each heart cycle. This discharge is coincident with the rapid rise in arterial pressure revealed by the carotid pulse curve. At high blood pressures this discharge becomes continuous, an effect which is likewise produced as the result of asphyxia. The general character of these discharges from the carotid sinus agrees closely with those found in the cardiac depressor nerve. The activity of the sensory nerve endings in both the arch of the aorta and the carotid sinus appears to constitute an important reflex control mechanism in preventing excessively high blood pressures or dangerously high heart rates.

Cushing[3] has reported new and fundamental observations on the effects of pituitrin and pilocarpine injected into the cerebral ventricles. He states that the active principle of the neurohypophysis is secreted in part into the blood stream and in part into the cerebrospinal fluid. As is already well known, this hormone from the posterior half of the gland, when injected into the blood causes vasoconstriction and stimulation of the muscle in the lower bowel. When introduced into the ventricle, on the contrary, Cushing found that it causes flushing, sweating, salivation, vomiting, and a pronounced fall in body temperature. An intravenous injection resembles a sympathetic discharge, while the intraventricular injection appears to produce a cranial autonomic or parasympathetic effect. Pilocarpine introduced into the ventricle gives a very similar response. As a working concept it may be assumed that under emotional stimuli the posterior pituitary becomes discharged through nerves running up into the region of the diencephalon, that the active principle diffuses into the cerebrospinal fluid, and thence by further diffusions acts upon the diencephalic nuclei of the parasympathetic system. When the autonomic centres in the dien-

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cephalon are infiltrated by tumors, this reaction fails to take place. The effect produced on the parasympathetic system is as diffuse in its character as the reaction of the sympathetic to adrenalin. Cushing feels that the neurohypophysis may bear somewhat the same relation to the parasympathetic division that the adrenal medulla does to the sympathetic division of the autonomic nervous system.

Ken Kuré and his collaborators in Japan[4] have studied the vagus and sympathetic fibres histologically. An important outcome of their work is to show the mixed character of the splanchnic and vagus nerves running to the stomach and intestine. Kuré has shown the presence in the posterior spinal roots of large numbers of finely myelinated fibres which do not degenerate centrally after the posterior root is cut. He believes that these are parasympathetic fibres which cause vasodilatation and excitation of the gastro-intestinal tract. Their excitatory action may be greater than the inhibition of the sympathetic fibres, so that stimulation of the splanchnic trunk may provoke contraction, peristalsis, and vasodilatation of the corresponding region of the intestine. Nicotine application to the sympathetic ganglia blocks all the inhibiting sympathetic fibres and permits the parasympathetic nerves to cause unopposed contraction with an increase of peristalsis and blood flow to the intestines. In a similar manner the vagus nerve receives many sympathetic fibres in the region of the superior cervical sympathetic ganglia and other less important anastomoses throughout its length. These observations are important as they show why observers of the past have obtained such confusing results on stimulating either the vagus or the splanchnic nerves. Neither one of them gives a clear-cut stimulating or inhibiting effect on the bowel, as both are mixed nerves.

Tower[5] casts a considerable degree of doubt on that part of Kuré's histological work identifying the thinly myelinated fibres which remain in the cut posterior roots as parasympathetic efferent pathways. By the use of serial sections, she has been able to demonstrate that these thinly myelinated fibres are growing proximally from the injured anterior root. She concludes that it is quite obvious that Kuré was dealing with a condition studied by Lugaro in 1906—the invasion of the sensory path of the proximal dorsal root by motor fibres from the ventral root. It is doubtful if any of these fibres succeed in penetrating through the sclerosed posterior root region into the deeper normal parts of the spinal cord. This work does not invalidate Kuré's physiological observations, that stimulation of the splanchnic nerves, after their sympathetic synapses have been blocked by nicotine, causes vasodilatation in the intestinal vessels and an increase in peristalsis—a parasympathetic response.

As an outcome of the studies on the total removal of the sympathetic system from animals which have been made in the physiological laboratories at Harvard, Bradford Cannon[6] has shown how little in the way of permanent effect on blood pressure is produced by the removal of the sympathetic system. After removal of the lumbar sympathetic chains and severance of the right splanchnics and again after removal of the right thoracic chain from dogs, the blood pressure undergoes an initial fall which is followed promptly by a rise reaching considerably above normal before it slowly returns to its previous range. After the complete exclusion of sympathetic control (by resection of the left thoracic sympathetic chain and the left splanchnics), the blood pressure drops lower than after the previous operations, but then returns to about its normal level. This shows the futility of attempting to treat hypertension by operation on the sympathetic nervous system. Many such attempts have been made in the past, but all without success. Garrey and Butler[7] studied these dogs after total sympathetic removal and showed that severe exercise, carbon dioxide and heat do not cause the usual increase in leucocytes found in normal dogs or in man. Under these circumstances the characteristic increase in red count which occurs in severe exercise also fails to appear.

During the past year there has been considerable discussion concerning the permanent effects produced by various types of sympathetic operations. Two important pieces of evidence have been brought forward to show that sympathetic nerves can regenerate. Brücke[8] cut the cervical sympathetic trunk proximal to the superior cervical ganglion and investigated the regeneration of the fibres. In six days he began to notice microscopic regeneration of the fibres which advanced about 1 mm. per day. In eighteen days one thousand nerve fibres had crossed the point of suture. In twenty-six days the pupil was capable of complete dilatation, and in twenty-eight days the tonus of the peripheral vessels of the ears had entirely returned. One hundred and twenty days after operation regeneration appeared to be complete.

Tower and Richter[9] investigated this same problem by a different method. They did this by measuring electrical resistances of the skin. The skin has a fairly constant electrical resistance which may show a sudden marked reduction accompanying emotional activity or painful stimulation. This reaction is probably due to increased activity on the part of the sweat glands, which are innervated by the sympathetic nerves. It was shown by experiment on cats and monkeys that these functions depend on an intact sympathetic innervation. The preganglionic sympathetic fibres were cut on the right side before entering the stellate ganglion in a series of twenty-six adult cats. Records of electrical skin resistance of the forepaws and observations

on sweating were also made. The immediate effect of operation was an increase in skin resistance, which reached a peak many times the normal figure between the second and fifth postoperative days. Between the third and seventh weeks skin resistance again approached the normal figure. A return to normal activity averaged twenty-eight days.

II. Diagnostic Tests

The discovery that certain general and local anesthetics paralyze the sympathetic nervous system and the utilization of this principle to demonstrate the degree of vasodilatation or the relief of pain which can be brought about by sympathectomy were described in last year's review. During the intervening period numerous further articles have appeared on the subject of diagnostic nerve blocking with procaine. Morton and Scott[10] now recommend the injection of the mixed peripheral nerves instead of spinal anesthesia or the paravertebral injection of the sympathetic ganglia. In the case of the foot they recommend blocking the posterior tibial nerve at the ankle, as this gives complete vasodilatation in the anesthetized sole and tips of the toes and the technique is so simple that it can be carried out in the office. These writers also define the normal "vaso-dilatation level" as the maximum surface temperature which can be produced after complete paralysis of the regional vasoconstrictor nerves in a normal limb. In the case of the tips of the fingers and toes, this is at about 88° to 90° Fahrenheit; as the normal temperature in this region is considerably below that of the thigh or trunk, the rise in temperature after novocaine block is much greater in the phalanges than in the upper portions of the arm or leg. This idea of a normal vasodilatation level is an important contribution as it establishes a base line to which the response in various types of peripheral vascular disease can be compared. In Raynaud's disease the elevation of temperature should reach the normal vasodilatation level, whereas in thromboangiitis obliterans and arteriosclerotic gangrene it is distinctly less.

De Takats[11], Livingston[12], and Swift and Flothow[13] have all written enthusiastically about the use of local anesthesia as a preoperative test for the value of sympathectomy. As the last two writers justly state, "this diagnostic method has completely done away with the hit or miss type of sympathetic surgery which has been so prevalent heretofore. It is no longer necessary to do experimental sympathetic surgery."

Lewis and Pickering have developed a new and very practical method for differentiating vasospasm and arterial occlusion, which is described below under Raynaud's disease.

III. Operative Methods

A. *Periarterial Sympathectomy*

Very few articles, either for or against this much debated operation, have appeared during the past year. Colp and Mage[14] have used periarterial sympathectomy in seventeen cases of lower extremity fracture. In seven recent fractures they claim a reduction of time in obtaining clinical union of eleven days and no tendency in any of these cases toward delayed union. In a further series of ten cases of delayed union of fractures of the lower extremities which were ununited after an average period of seventy days, clinical union resulted twenty-one days after periarterial sympathectomy in eight cases. They conclude that experimental and clinical evidence seem to justify further trial of this operation in recent fractures and in those with non-union.

Rogers[15] mentions the transitory nature of periarterial sympathectomy in experimental animals. In dogs, cats, and rabbits the increase in temperature lasted only a few hours to a few days. He reports a case in man where this operation was done for ulcers in an old burned area. Here the skin of the operated leg felt somewhat warmer than the unoperated side for a period of six weeks, but as in so many of these reports no definite temperatures are recorded. Doppler[16] has written an extensive summary of periarterial destruction of the sympathetic nerves by his method of painting with 7 per cent solution of carbolic acid. He feels that this produces a reflex depression of sympathetic tonus.

B. *Sympathetic Ganglionectomy*

During the past year Adson[17] has modified his technique of cervicothoracic ganglionectomy to the extent that he now recommends removal of the first, instead of the second rib, and specifies that the entire inferior cervical sympathetic ganglion must be removed, as well as the first and second thoracic. By the previous second rib operation Adson claims that although he removed what he thought to be the entire cervicothoracic ganglion, yet he often observed an incomplete dilatation of the retinal arteries, which made it apparent that sympathetic fibres were entering the trunk above the limits of his operation. He also observed that when the lower cervical and upper thoracic ganglia were not fused into a single structure, the second rib operation frequently failed to obtain complete vasorelaxation.

The same difficulties with the second rib operation have been noted in the Peripheral Vascular Clinic of the Massachusetts General Hospital where many of the cases operated through the lower approach have failed to maintain complete vasomotor paralysis. It is still an open question whether the majority of cases of Ray-

naud's disease of the upper extremities can be permanently relieved by the first rib operations now recommended by Adson.

Wagener[18] of the Mayo Clinic reports interesting observations on the retinal circulation after Adson's operations. Out of thirty-five patients on whom bilateral ganglionectomy was done, dilatation of the retinal arteries occurred in one or both sides in thirty-four and of the veins in twenty-five. Complete absence of dilatation was noted in only one case, which was a patient with Buerger's disease of long duration. Dilatation of the arteries occurs as frequently as narrowing of the pupils and orbital fissures and it has been seen to last for over a year. The irregular amount and distribution of the changes noted following these operations are probably due to the variable origin of the sympathetic fibres supplying the eye. The usual effect of such operations seems to be the production of incomplete rather than complete Horner's syndrome.

Harvey and his coworkers[19] in New Haven have described experimental observations which show the efficiency of sympathectomy in dilating the collateral blood supply to a limb after the ligation of the principal arteries. In the dog, ligation of the iliac arteries close to the aorta was always followed by a decrease of temperature in the corresponding foot varying from 10° to 30° Fahrenheit. In the majority of the experiments the temperature rose to the previous level about thirteen hours after ligation. If the lumbar sympathetic ganglia were removed at any time during this period the temperature of the foot returned rapidly to normal; if they were removed at the time the artery was ligated, no fall in temperature took place. Transection of the spinal cord at the first lumbar level had no effect on the temperature, which is in accord with Langley's original observations that the highest vasomotor fibres to the leg leave the cord in its lower thoracic segments. These observations should be of distinct practical value for surgeons who contemplate doing high ligations of the iliac vessels in the human being. Under these circumstances it would be very easy to remove the lumbar ganglia at the same time and thereby reduce the risk of gangrene.

C. *Alcohol Injection of Vasomotor Nerves*

A number of surgeons, notably Flothow[20], are beginning to attempt injection of the sympathetic rami and ganglia instead of operative excision for the relief of vasospasm. Flothow reports twelve cases of paravertebral lumbar alcohol injections for arteriosclerosis. These gave marked relief of pain in the feet, improvement of circulation, and healing of trophic lesions in ten. He recommends it preliminary to amputation in order to increase blood supply, to insure healing of the stump, and to avoid amputation stump neuralgia. A similar injection was done in one case of Raynaud's disease and the immediate result was reported to be as good as sympathectomy.

In three cases of chronic polyarthritis, paravertebral alcohol injection was performed on one side, ganglionectomy on the other. Relief of pain and improvement of joint function on the two sides were equal. The sides operated upon, however, maintained their increased temperature better and the patients preferred operation to injection on account of the troublesome neuritis which follows so many of the alcohol injections.

IV. Effects of Sympathectomy on the Circulation

A. *Raynaud's Disease*

Lewis and Landis[21] have brought out further evidence in favor of the theory that Raynaud's disease is due to a local vascular defect, rather than to spasm set up by a hyperactive vasomotor nervous system. They claim that in the milder forms of the disease this defect is expressed as a susceptibility of the digital arteries to enter a state of spasm. This can be done by local chilling of the base of the fingers or, at times when the body is exposed in a cool room, by a vasoconstrictor reflex of normal intensity. In the severer forms, spasm is reinforced by local structural changes which are apparent on microscopic examination. They reëmphasize the fact that there is no abnormal increase in vasoconstrictor tone. There is a form of diffuse scleroderma in which those portions of the skin which are exposed to cold are commonly involved. In these cases they believe that it is not deficient blood flow which leads to the atrophy and scarring, because scleroderma of the face can develop without any previous evidence of deficient circulation.

Lewis and Pickering[22] describe a new method of studying changes in the peripheral circulation. They place the patient in an electrically heated chamber with the hands protruding and exposed to the temperature of the room. In this way the body can be warmed while the hands are exposed to the cool temperature of the room. This warm chamber is valuable clinically in differentiating obstruction arising from spasm and structural disease of the arteries. In the former, the rise in temperature of the hands is rapid when the body alone is warmed. In the latter, the response in the hands is slow and less pronounced. The chamber can also be used in determining when the sympathetic innervation of the vessels has been lost, as under these circumstances the temperature of the denervated fingers rises very slowly. The response is effected entirely through sympathetic nerves and the authors postulate that these nerves must contain vasodilator, as well as vasoconstrictor fibres.

Boggon[23] has applied Lewis' methods to the study of Raynaud's disease and agrees that novocainization of the ulnar nerve does not give complete relief from vasospasm in the little finger. He goes on to say that on local cooling of the palmar vessels in Raynaud's disease the arterial lumen may be contracted down to obliteration and the fingers become blue. After sympathectomy, however, although local spasm still occurs, it acts on vessels of a larger calibre, so that they still have sufficient diameter to allow the blood to reach the fingers. Thus, although it is argued that Raynaud's disease is due, not to a vasomotor neurosis but to a local lesion of the palmar vessels, the division of the sympathetic supply to the upper limb is strongly advocated for severe cases. By carrying out this operation, Boggon states that the patients are benefited, but not cured. The subsequent attacks are much less severe and less frequent than before and the patients are relieved both subjectively and objectively.

The writer[24] has summarized the results of sympathectomy in thirty-five cases where this operation had been employed in the Peripheral Vascular Clinic of the Massachusetts General Hospital. Four of these were typical of Raynaud's disease. The methods of diagnostic novocaine injection utilized in this clinic to determine the degree of vasodilatation which can be produced by sympathectomy are described. We have consistently maintained that the sympathetic ganglia should be blocked paravertebrally and have not used Lewis' method of blocking the ulnar nerve alone. As the result we have obtained much greater rises in temperature of the fingers than those reported by Lewis and it has been quite impossible to produce evidence of local spasm after these injections. The same was true after sympathectomy in three out of the four cases which at the time of this report had been followed from five to eight months.* The fourth case showed a severe form of the disease with extensive scarring of the tissues which permitted palliation, but prevented complete relief by operation.

Simpson, Brown, and Adson[25] also believe in Raynaud's view of a vasomotor neurosis as the etiology of this disease. They criticize Lewis' evidence on account of the limitation of his essential experiments to severe and complicated types of the disease. In the characteristic but uncomplicated cases complete interruption of the sympathetic nerve fibres by local anesthetization or by surgical operation prevents any manifestation of local spasm. It is important to note that they also do not depend on simple infiltration of the ulnar nerve to block all the vasoconstrictor fibres to the little finger. To do this Lundy recommends a local block in the palm of the hand at the base of the little finger as well as injection of the ulnar nerve, as he feels that other vasoconstrictor pathways may reach the little finger from the median nerve. Cervicothoracic sympathetic ganglionectomy occasionally fails to give complete relief because of failure to section all of the sympathetic fibres. In the author's experience lumbar sympathetic ganglionectomy is always completely successful. Lewis, it should be pointed out, has never reported the results of his tests in Raynaud's disease after operation in the lower extremity. He also has never explained the excessive sweating which takes place in the majority of these cases and which is known to be produced by increased irritability of the sympathetic nervous system. The great preponderance of the experimental evidence brought out at the Mayo Clinic confirms Raynaud's original view of an abnormality of the sympathetic nervous system.

*More recent examination shows a recurrence of vasospasm in two of these cases. In each instance there has been a slow, definite reëstablishment of sympathetic nerve activity.

B. *Other Conditions Associated with Vasospasm*

There is no question but that ganglionectomy can arrest the advance or even bring about some regression of fibrosis in the type of scleroderma which often accompanies Raynaud's disease and is limited to the extremities, when diagnostic novocaine injection shows that a satisfactory degree of vascular dilatation is possible[24]. It cannot help the idiopathic type of scleroderma which involves the face and large areas of the body. This operation is also of great value in certain cases of chronic ulceration in the extremities. Cases of cure of long-standing ulcers in erythema induratum are recorded by Homans and from our clinic[24], also of trophic ulcers following poliomyelitis and in other neurogenic conditions by Woltman, Allen, and Craig[26].

Enthusiastic reports of the benefit following sympathetic ganglionectomy in favorable cases of polyarthritis continue to emanate from the Mayo Clinic[27], from Flothow[20], and Moore[28] (a physician who was operated on for this condition at the Mayo Clinic). At the Massachusetts General Hospital there have been two extremely good results of this operation in cases of traumatic arthritis and two complete failures in the chronic infectious type of the disease in the hands.

V. Effects of Sympathectomy on Pain

A. *Migraine*

A new application of sympathetic surgery has been made by Dandy[29] to migraine. Reasoning that in this affection the pain covers a larger area than can be accounted for on the basis of any cerebrospinal nerve distribution, Dandy removed the cranial sympathetic outflow by resecting the cervicothoracic ganglion in two cases. Both of these remained completely relieved up to the time of publication (two and one-half

to six and one-half months after operation). Dandy further points out that it is important to do a complete operation by the posterior approach, as he failed to benefit one of these patients by a preliminary removal of the superior cervical ganglion.

B. *Cardiovascular Pain*

Burget and Livingston[30] investigated the pathways of pain from the peripheral arteries. They found that injection of five per cent lactic acid into the brachial artery of the dog causes painful afferent impulses and claim that these arise in the walls of the vessels rather than from the tissues supplied by the artery. They found that they are unaffected by paralyzing all the sympathetic innervation of the vessel through the removal of the stellate and second thoracic ganglia. The pain impulses appeared to enter the cord by the seventh and eighth cervical and the first dorsal posterior roots.

Sutton[31], in reviewing the theories of cardiac pain, quotes his experiments on dogs which were reported in last year's review and adds to them further observations on monkeys and clinical observations on man. In animals pricking or pinching of the parietal pericardium elicits a pain response, stretching or pulling does not. Neither a mechanical nor a chemical irritation of the visceral pericardium evokes any pain. Temporary closure of either a coronary artery or vein invariably elicits a pain response. This is true of both the right and left coronaries and their branches, the severity of the pain varying with the amount of the circulation cut off. The pathway of the pain is through nerve fibres that accompany the coronary arteries and pass to the central nervous system through the left sympathetic chain of ganglions. Pain can also be produced by passing a probe down the carotid artery and inserting it into a coronary orifice, but stretching of either the normal aorta or the aortic ring, on the other hand, fails to produce pain. Preliminary observations on stretching an aorta which has been previously inflamed also indicate that pain does not occur. It is, therefore, evident that pain occurs only when the blood flow to the myocardium is diminished or stopped. This is the one mechanism which appears to give a reasonable explanation for all the varied pathological conditions which cause angina pectoris in the human being.

Leriche, Herrmann, and Fontaine[32] have shown that ligature of the vertical branch of the left coronary artery is followed by rapid death. In four dogs which had previously been subjected to resection of the sympathetic cardiac nerves, death occurred in only one case. A second dog died eight days later of sepsis, and two remained in good condition. These animals responded well to exercise a year after operation.

These observations are difficult to explain in view of Anrep and Segall's observations that only dilator nerves to the coronary arteries run in the sympathetic system. This has been confirmed during the past year by Danielopolu, Marcou, and Proca[33], and also by Greene[34]. Danielopolu, in opposition to Leriche, claims that removal of the cardiac sympathetic ganglia will cause actual injury to the heart and that the important stellate ganglia should not be removed in operations for angina pectoris.

Averbuck and Rachmilewitz[35] have checked up Danielopolu's observations that stellate ganglionectomy causes serious damage to the heart. The latter found that while a normal dog can stand bilateral removal of the stellate ganglia, a dog with two small branches of the left coronary ligated cannot. These authors were unable to verify Danielopolu's findings. While the heart rate fell moderately after removal of the ganglia, the heart tone, the force of its beat, and the blood pressure did not alter. The dogs showed unaltered cardiographic tracings after this procedure. All the work done in this country as well as in Leriche's clinic demonstrates that the efficiency of the normal heart, and even of the heart in angina pectoris, is not visibly reduced after resection of the stellate ganglia. This is probably due to the fact that many accelerator fibers reach the cardiac plexus directly from the second, third and fourth thoracic sympathetic ganglia.

C. *Pain from the Abdominal Viscera*

Learmonth has published some excellent papers on the innervation of the bladder with a description of the surgical anatomy of the superior and inferior hypogastric plexuses[36] and a most interesting physiological study of these nerves in the human being[37]. By studying the reactions of the exstrophic bladder to adrenaline and by cystoscopic observation during operations under spinal anesthesia in which the presacral fibres were first stimulated electrically and then cut, it was possible to work out the action of these nerves with great thoroughness, not only on the bladder, but on the ureteral orifices, the internal sphincter, and the prostate and vesicles as well.

Learmonth[38] has made a preliminary report on the results of operation on the hypogastric plexuses in bladder pain. In five cases of interstitial cystitis resection of the superior hypogastric plexus, (presacral neurectomy) gave subjective relief in all but one instance, as well as an objective improvement in the cystoscopic appearance of the bladder due to the resulting increase in its blood supply. Pieri has stated that these results can be improved by combining section of the presacral nerves with division of the lumbar sympathetic chains at the level of the sacral promontory. Learmonth, however, does not believe that severe vesical pain can be entirely controlled without removing the parasympathetic fibres which emerge from the sacral

foramina in addition to the presacral plexus. Such a resection of the inferior hypogastric plexus is a difficult and very radical procedure, as after the sacral divisions of the autonomic system are cut the emptying power of the bladder is paralyzed, forcing the patient to lead a catheter existence. This, however, is a small price to pay in certain cases of unbearable pain where the patient would be forced to endure continuous bladder drainage by catheter or cystostomy as the only alternative to cordotomy. Learmonth reports its use in two cases; one of malignant disease of the base of the bladder, prostate, and posterior urethra, the other in a patient with tuberculous ulceration of the bladder which failed to heal after nephrectomy. In each instance there was complete relief from all forms of vesical distress.

Learmonth[39] has also successfully employed resection of the superior hypogastric plexus in the treatment of bladder paralysis. He points out the fact that the sacral autonomic fibres, "the bladder emptying nerves", can be injured in spina bifida or in other injuries to the spinal cord in the lumbar region, while the sympathetic or "bladder filling nerves" descend in the lumbar trunks from a higher level and, remaining in unopposed control, may cause chronic retention of urine. After presacral sympathectomy, the inhibiting factor is removed and the patient can relax his voluntary external sphincter and empty his bladder by compression of his abdominal muscles.

Bauer, Salter, and Aub[40] report a useful method for the temporary relief of all forms of pain caused by colic of the hollow viscera. They recommend a slow intravenous administration of 20 cc. of sterile solution of five per cent calcium chloride, which promptly relieves the severe colic caused by lead or ureteral or biliary calculi. The physiological action of this drug lies in relaxing the smooth musculature composing the walls of these viscera, but whether this is due to any direct effect on the autonomic nerves which regulate their activity and transmit their sensations is unknown.

VI. Effects of Sympathectomy on Spastic Conditions

Very little has been said in behalf of this procedure during the past year and it appears to be rapidly joining the great multitude of operations which are greeted with great enthusiasm, run a short course of declining popularity, and then sink over the surgical horizon. Bisgard[41] and Carrell[42] have both essayed to repeat Royle and Hunter's experiments, but unsuccessfully or with conflicting results. They conclude that the operation has no direct effect in diminishing muscle hypertonicity. What little benefit it occasionally produces may be ascribed to such indirect reactions as altering the blood supply of striated muscle or its fatigability.

In closing this review it is of interest to note the publication of a new surgical monograph on the autonomic nervous system. It is written in German by Erich Hesse[43], professor of surgery in Leningrad and comprises 450 pages. The book gives a complete review of the anatomy and physiology of the autonomic nervous system and the surgical applications of this knowledge. The author has assembled all the investigations in this field (up to 1929), which have hitherto been scattered through the various European and American medical journals, and makes them available for the first time in a single volume. The illustrations are good, the bibliography excellent. Perhaps the only criticism that can be made of this book is that it is too complete, so that numerous unimportant or obsolete procedures are included among the most valuable methods in this branch of neurosurgery.

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THE ROLL CALL OF THE AMERICAN RED CROSS

The American Red Cross appreciates the fact that over a considerable period of years it has had tremendously valuable support from the nation's publications in the conduct of its Roll Call, held each year from Armistice Day to Thanksgiving, for enrollment of members for the following year. It anticipates with pleasure a continuance of the friendly relations built up for so long a period.

Your American Red Cross is now entered upon its second half-century of service. It has had the unfailing confidence and support of the people through the present testing period of world-crisis, and this support has enabled it in the past year to aid more than 10 million people suffering because of tornado, flood, drought and other disasters, and the added burden of unemployment.

It has administered successfully one of the most difficult tasks it has ever had, the distribution throughout all the states of 40 million bushels of wheat in the form of flour and feed for live-stock. This work has been accomplished at no cost to the beneficiaries, no profit to anyone.

Approximately 2,700 Red Cross chapters have taken part in unemployment relief and in addition the Red Cross has maintained its established services.

The extraordinary demands of the past year have, however, seriously depleted Red Cross resources. They must be replenished if the Red Cross is to continue to provide the service the people expect of it.

In its appeal for membership support this year the Red Cross must look to you for even more generous assistance than it has had in the past.

RESTRICTING FOR THE TIME BEING THE INTRODUCTION OF PARROTS INTO THE UNITED STATES

Whereas there have been officially reported in widely separated portions of the United States since the middle of December, 1929, a considerable number of human cases, some of them fatal, of a disease communicated by infected parrots; and

Whereas there is evidence that such parrots have been introduced from ports outside of the continental United States; and

Whereas there exists danger of further such introduction;

Therefore in order to prevent the further introduction of disease communicable from parrots to human beings, from ports outside of the continental United States into the United States, by virtue of the authority vested in me by section 7 of the act of Congress approved February 15, 1893, entitled, "An act granting additional quarantine powers and imposing additional duties upon the Marine-Hospital Service," it is ordered that no parrots may be introduced into the United States or any of its possessions or dependencies from any foreign port, for such period of time as may be deemed necessary, except under such conditions as may be prescribed by the Secretary of the Treasury.

This order shall take effect from and after this date. HERBERT HOOVER.

The White House,

January 24, 1930.

Executive Order (No. 5264)

This order was revised October 6, 1932.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 18441

HEART FAILURE OF MIXED ETIOLOGY

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. F. DENNETTE ADAMS: This was a private patient of Dr. B. H. Ragle whom both of us followed for eight years. She was seen first in May, 1923, at the age of fifty-three.

The family history is not important. Her husband was living and well and she had one son living and well.

She had the usual children's diseases, including questionable mild diphtheria at twelve, scarlet fever at fifteen and questionable mild pleurisy. She originally gave a history of having had a few fleeting rheumatic pains at the age of twenty when in college, but later it was brought out that at this time she had been ill for several months with very definite rheumatic fever. Her tonsils were taken out at forty-five. There was no operative history except a Cesarean section for the living child, her one previous pregnancy having been attended by loss of the child at birth because of some obstetrical complication.

She had been perfectly well and active between the ages of thirty and forty, although at about the age of forty she developed asthma which we believed, as time went on, was based on bacterial allergy rather than some extrinsic cause. She was extremely susceptible to upper respiratory infections; we followed her through several definite attacks of bronchopneumonia, all of them starting as acute upper respiratory infection and followed by prolonged asthma of several weeks' duration.

At the age of forty-two, while living in Texas, she developed anesthesia in the right arm and shoulder which persisted until death. She was thoroughly studied in Texas at that time. One doctor performed a blood test (presumably a Wassermann) and then gave her several injections of a yellow fluid (presumably salvarsan). We performed several Wassermanns at various times; all were negative. Spinal fluid examination was normal. Several neurologists saw her after she came under our care, but made no definite diagnosis and found no cause to account for this symptom. It gave her a great deal of trouble.

Except for a blood pressure of 160/100 when we first saw her in 1923 nothing of importance was found in the cardiorenal system. She was examined at frequent intervals from 1923 until 1929, chiefly with reference to the areas of anesthesia and the upper respiratory symptoms.

In February, 1929, she was seized in the middle of the night with severe pain, a feeling of compression in the chest and dyspnea. We were constantly with her for twelve hours and we were both quite convinced that she had had an attack of coronary thrombosis. She developed moderate fever for a few days, a leukocyte count of 15,000 and a suggestion of precordial rub, but one could not be certain that the sound suggesting the latter was not a soft diastolic murmur. Dr. W. H. Smith saw her on several occasions during this episode. From that time (February, 1929) until a few months before her death (1931) she had changing murmurs; but just what type of valvular lesion, if any, was not clear. In the last few months, however, the typical presystolic rumble of mitral stenosis appeared and remained fairly constant, although obscured at times by variation in her blood pressure, which ranged from 200/100 to 140/70.

In the summer of 1929 while at a summer hotel in Maine she went through another episode which we were unable to explain. She suddenly developed coma, high fever, and a high white count. She was transferred to the Phillips House and remained there eight weeks. She was investigated thoroughly but no diagnosis was made. We were not sure that she did not have encephalitis.

Later in 1930 the heart began to fibrillate. Following this she developed frequent attacks of cardiac decompensation with marked dyspnea which necessitated increased restriction of her activity. The changes in the murmurs became more frequent and confusing, but there was usually a presystolic rumble. The blood pressure fluctuated also. She was practically confined to bed and chair because of congestive failure. There were no anginal attacks.

In 1931, seven years after we had first seen her, when she had been relatively well for the past month with no essential change, she suddenly developed rapidly increasing dyspnea. At that time the blood pressure was 140/90. Three or four days later it was 100/80. The liver, which was always a little enlarged, had become definitely more so and more tender. The following night she suddenly became decidedly worse, with a sense of suffocation, precordial tightness and even more dyspnea. In another hour she was moribund and pulseless. It was impossible, of course, to hear her heart because of noisy respiration. Definite edema of the lungs could not be made out. She rapidly failed that night and died the next morning, forty-eight hours after the onset of the sudden change.

Clinical Discussion

Dr. Adams: We believed we were dealing with a case of rheumatic heart disease, probably pure mitral stenosis; an old infarct due to previous coronary damage; arteriosclerotic and hypertensive heart disease; chronic bronchitis, emphysema and bronchiectasis. We were never able to diagnose the condition giving rise to the anesthesia of the arm and shoulder. We suspected that the sudden terminal coma was due to embolism from a thrombosis either in the left auricle or in the left ventricle overlying the site of the old infarct; possibly cerebral thrombosis and hemorrhage. It seemed unlikely that cardiac failure alone could produce coma so rapid in its onset.

Dr. F. D. Adams and Dr. B. H. Ragle's Diagnoses

Coronary thrombosis, old.
Probable old infarct of the heart.
Auricular fibrillation.
Rheumatic heart disease with mitral stenosis.
Arteriosclerotic heart disease.
Thrombus in the left auricle or ventricle.
Cerebral embolism?

Anatomic Diagnoses

Rheumatic heart disease with acute and chronic endocarditis of the mitral and aortic valves with mitral stenosis and aortic stenosis.
Chronic mural endocarditis of the left auricle with mural thrombus.
Coronary sclerosis and occlusion without infarction.
Fibrinous pericarditis.
Central necrosis of the liver.

Pathologic Discussion

Dr. Tracy B. Mallory: The postmortem examination was more or less of a surprise to everyone, because the one outstanding lesion was an extreme degree of mitral stenosis. The mitral valve, which was reduced to a very narrow slit, was markedly calcified. There was also a fairly definite though less marked aortic stenosis with interadherence and some calcification of the bases of the cusps. She did have also marked coronary disease. There were numerous patches of extreme narrowing, for instance one at the mouth of the right coronary. The circumflex branch of the left coronary showed only a moderate atheroma, but its descending branch was apparently occluded by an old atheromatous mass with no fresh thrombus. The circumflex branch of the right coronary about 4 centimeters beyond its origin also showed a marked area of narrowing, and there we found a small fresh thrombus just anterior to the most narrow spot. So I think that final thrombus was the immediate mechanism of death, shutting off an already much narrowed coronary. The closure of the left coronary was probably the definite attack she had two years before death. It had not left any gross infarction, but there was an adhesion to the pericardium over the apex of the left ventricle. The muscle there was not thin, but showed microscopically very numerous and extensive fibrous bands. In a good many cases of coronary occlusion no single large patch of complete necrosis occurs, but scattered areas of necrosis, each perhaps only a millimeter or two in width, are found separated by narrow bands of uninjured muscle cells. I think that probably at least 30 per cent of coronary thromboses, perhaps more, are followed by a rather patchy necrosis in the myocardium rather than by any single gross infarction. The mitral stenosis undoubtedly must have gone back to her rheumatic fever at the age of twenty. It seems astounding that she could have gone such a long period having so few symptoms and particularly so few signs as she showed.

Dr. Howard B. Sprague: Was the fresh thrombus proximal or distal to the coronary narrowing?

Dr. Mallory: Both.

Dr. William B. Breed: Of how much importance is the period of auricular fibrillation in this history as an indication of the etiology, Dr. White?

Dr. Paul D. White: In the case of a young or middle-aged patient one should be suspicious of mitral disease, but in an older person auricular fibrillation is not rare in hypertensive or coronary heart disease. I should think the note that Dr. Adams made of occasionally hearing a presystolic murmur, which might have been that of mitral stenosis, was the chief clue to the diagnosis in addition to the rheumatic history. Her circulation was so weak that the murmur might have escaped notice. I suspect however that when she was in better health she should have had enough of a mitral diastolic murmur to warrant a diagnosis of mitral stenosis.

Dr. Adams: We saw her right along for six years before the angina or coronary episode. If there was a mitral diastolic, three different observers missed it.

Dr. White: But you commented on the fact that a presystolic murmur was heard several times.

Dr. Adams: That was after the coronary episode, when we began to try to determine what the murmur was that we were quite sure had not been there before.

Dr. Sprague: What did the electrocardiogram show? Did it ever show right axis deviation?

Dr. Adams: There was no right sided axis deviation; there was auricular fibrillation; T_1 and T_2 were diphasic, with chief deflection downward and ST interval below the base line; T_3 was inverted.

Dr. White: How much aortic stenosis was there?

DR. MALLORY: It was definite though relatively mild.

This case to some extent answers the question as to how she could have gone thirty years with no suspicion of mitral disease, and to the very end it is questionable whether any of her symptoms were produced by it.

DR. WHITE: She lived a very quiet life, didn't she? Had she ever been very active?

DR. ADAMS: She had been active to the age of forty. After that she was less active, but it was apparently her asthma which slowed her up.

DR. WHITE: I have seen marked mitral stenosis and fibrillation in a woman of sixty-five who had lived very much of an invalid life for twenty years or so. That kind of existence was probably the chief reason why she lived so long. Another very instructive patient of mine is a man now over seventy years of age, in very good health in spite of well marked mitral stenosis and auricular fibrillation. He had chorea at the age of eight years.

DR. MALLORY: We also have in our series one very interesting case that had been very carefully followed for nearly forty years. This was a woman who at seventeen had acute apical pulmonary tuberculosis and was sent to Saranac. A year and a half later she had rheumatic fever. During her convalescence from that a definite murmur of mitral stenosis was heard. The following year she married, and later she had five children. She eventually died of lymphatic leukemia without ever having had any cardiac symptoms, with a mitral valve that would barely admit the entrance of one finger.

DR. WHITE: How old was she when she died?

DR. MALLORY: Fifty-eight.

FURTHER DISCUSSION OF CASE 18131

Published March 31, 1932

DR. HOWARD B. SPRAGUE: This was a very obscure case and one's diagnosis depended on one's point of view on that particular day. He had a history of paroxysmal dyspnea at night; he had lues; he had heart failure with normal rhythm and he had aortic regurgitation. The findings at the apex of the heart, as you heard, were variable. He had a systolic murmur which might I believe have been an Austin Flint murmur from ventricular dilatation. He did not have a sharp first sound.

The joker in the straight luetic picture is that he was said to have had heart disease for twelve years, and in addition he had well marked right axis deviation by electrocardiogram. My first note stated that I favored a diagnosis of syphilitic heart disease. I questioned whether he had in addition rheumatic heart disease, but I doubted it. I had difficulty with the right axis deviation, and I thought it would be well to fluoroscope him myself in spite of having the diagnosis of the X-ray Department, which was that he had rheumatic heart disease. So Dr. White and I fluoroscoped him.

That really made it even more difficult, because he did have definite evidence of increased pulmonary pressure and that would fit in with some disease of the mitral valve, although we questioned at that time a possible luetic sclerosis of the pulmonary tree with increased pressure from that cause.

The second time he came in Dr. White saw him. He favored rheumatic heart disease, perhaps with luetic aortitis also. So in the first case I thought the main trouble was lues with perhaps some rheumatism, while Dr. White thought the main trouble was rheumatism with perhaps some lues. The Pathologic Department is the only one that really knows.

DR. GEORGE W. HOLMES: I should like to read the x-ray interpretation. "The appearance is that of a large heart with passive congestion, *probably* rheumatic." We did not say positively that it was rheumatic. There is no doubt that the heart is enlarged as shown by the x-rays, but I think there is some question as to whether the right or the left ventricle is the one enlarged. We have no notes here to help us decide that question. The supracardiac enlargement is less than one would expect in long standing luetic aortitis with aortic regurgitation. The findings are more suggestive of rheumatic than of luetic heart disease.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: At autopsy we found a very unusual heart in some respects. I have never seen one quite like it. We felt quite sure however that he had both syphilitic and rheumatic heart disease. The heart was moderately enlarged, weighing 550 grams. It showed very marked dilatation of its right side, the apex of the right ventricle actually forming for most of the hypertrophy that was visible in the x-rays. There was definite separation of the cusps of the aortic valve and there were some unusual changes in the aorta which consisted both of areas of scarring and of some peculiar raised white plaques which stood up from the aorta 2 millimeters in their fresh condition. They were white, not yellow like ordinary atheromatous plaques. The entire intima of the aorta seemed considerably thickened. All the valves showed fresh little warty vegetations typical of acute rheumatic endocarditis. The mitral valve showed considerable thickening of the chordae tendineae and some thickening of its margins as well as the fresh vegetations. The tricuspid valve measured 14.5 centimeters at the base and we thought that there was probably a relative tricuspid regurgitation there,

though the lesions on the valve itself were only the acute granules; there was no chronic rheumatic lesion there. The coronaries were essentially negative.

The most peculiar thing of all was an endocarditis of the left ventricle just below the aortic valve. There was a patch on the surface of the left ventricle running up slightly to one of the cusps, which was fiery red, definitely thickened and slightly granular. It seemed like a definite endocarditis of the left ventricle itself, just below the aortic valve. Endocarditis in the auricles spreading up from the mitral valve is relatively common, but it is rare to see endocarditis in the ventricle spreading down from the aortic valve. I have seen healed stages of such a process, but never caught one in the acute stage before.

The aortic lesions turned out to be a combination of lues and arteriosclerosis. Their peculiar appearance was due to very marked fibrous thickening of the intima one to two millimeters in width overlying the atheromatous deposits. In other areas there was typical luetic medial scarring. Now that the specimen has been fixed in formalin the lesions do not stand out nearly so prominently and the yellow shows through, so that it is much more obvious at the present time that it is an atheromatous lesion.

On account of the great variability in his metabolic rate the question of hyperthyroidism was raised at one time. Autopsy showed a small thyroid with pretty definite chronic thyroiditis but no epithelial hyperplasia, the type of thyroid one would expect to be associated with a low rather than an elevated basal metabolism.

Dr. Franklin G. Balch, Jr.: I should like to ask Dr. Sprague what the prognosis of these two types would be.

Dr. Sprague: I think that when a patient with luetic heart disease begins to have failure the prognosis is very bad, usually less than two years. On the other hand with a rheumatic heart several bouts of congestive failure can be survived and the patient may live for several years afterwards.

Dr. Holmes: Was there any actual dilatation of the aorta?

Dr. Mallory: No, I should say not.

Dr. Paul D. White: How do you explain the enlarged right ventricle?

Dr. Mallory: I have no very good explanation for it.

Dr. White: The electrocardiogram pointed to right ventricular enlargement. It showed well marked right axis deviation, which in the absence of considerable mitral valve disease is not easy to explain. Mitral regurgitation as well as mitral stenosis can cause strain and enlargement of the right ventricle, of course. Was there much of any mitral stenosis or regurgitation?

Dr. Mallory: I should say there was fairly definite regurgitation but not much stenosis. There was a fair amount of pulmonary arteriosclerosis, but nothing very striking, not nearly so much as many mitral cases would show.

I think this coincidence of luetic and rheumatic heart disease seems a little commoner than it is ordinarily given credit for being. We certainly have had a number of cases in the past two years in which we felt quite sure at autopsy that one type was superimposed upon the other.

Further Discussion of Case 18081

Published February 25, 1932

Dr. Tracy B. Mallory: It seems to be a fairly common thing for these cases with syphilitic narrowing of the coronary mouths to die during sleep as this man did. I know of two other similar cases.

Dr. Robert S. Palmer: Without thrombosis?

Dr. Mallory: Yes.

Dr. William D. Smith: Have you any explanation for that sudden death? I should think insufficient blood supply would occur during activity rather than in sleep.

Dr. Mallory: No; I cannot explain it. It seems fairly obvious that these patients must develop rather extensive and for a long time pretty adequate collateral circulation, but that that collateral may fail suddenly.

Dr. J. H. Means: Don't you think that circulatory reactions occur during sleep? Crises of high blood pressure tension have been observed to occur during sleep, and in my own experience I have known of persons waking up with the heart beating violently. It is a common story in hypertensives. I think sudden death with the coronaries narrowed but not thrombosed is not uncommon. I can think of one other case which you autopsied here where there was fairly sudden death, not during sleep, and the only finding was narrowing of the coronaries without a sign of thrombosis. It may be that given a person with relatively inadequate coronary circulation and superimposed on that some sort of crisis taking place during sleep, sudden death occurs.

Dr. Mallory: Certainly very real emotional disturbances can occur during sleep.

Dr. Means: They certainly can.

Dr. Mallory: I think there is very little doubt that this patient had a combination of two forms of heart disease, luetic and rheumatic, although the rheumatic was a very slight affair and presumably played no part whatever in the symptomatology.

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The Journal does not hold itself responsible for statements made by any contributor.

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A SYMPOSIUM ON ARTHRITIS

IT is most opportune that the Boston Orthopaedic Club has arranged a program on Arthritis for its first and second meetings this Fall. The menace to society as a whole, to industry, to our economic structure, and to the happiness of individual and family life from this malady is becoming increasingly apparent to the public and to the profession at large. It has been conservatively estimated that there are 145,000 arthritics in Massachusetts alone at this time. How well are we as a profession equipped to deal with this perplexing problem? How well informed is each one of us to meet adequately the problem of the patient who reports first to his physician with symptoms which definitely warn of an impending attack; or in the case of the patient upon whom the acute attack has already descended; or again with the patient who, discouraged, is confronted with the long, hard struggle against crippling disabilities? There are already unmistakable signs of general dissatisfaction among the laity and an increasing public enlightenment concerning the seriousness of the problem. A public demand for thoroughly informed and experienced medical attention is growing. How are we responding?

Arthritis demands, first of all, a sound understanding by every member of the profession of the nature of the malady, namely, that the joint manifestations are but symptoms of a constitutional disease, that this disease may only be overcome by dealing with it as a general, and not a purely local, depression. Secondly, the responsible physician must then be himself informed of sound methods of analysis whereby he may detect the various faults and how to correct them. Failing in these, he must recognize the necessity of placing his patient in other professional hands adequate to the important task.

A thorough review of the accredited body of knowledge of ARTHRITIS — clinical, laboratory, and social is to be presented in a symposium on the subject consisting of two meetings and a clinic by the Boston Orthopaedic Club. Progress in combating this disease, the need for its better understanding by the public and the profession and for evaluation of the immense amount of research in this field, and the pressing need for better treatment of the arthritic in all phases of his contact with the profession lead us to urge participation by the general practitioners of New England in the excellent program which the Boston Orthopaedic Club has arranged. Attention is called to the program on page 812 of this issue.

WHY INCREASE THE BUDGET NOW?

THE chiropractic referendum will be on the ballot on election day with a description of many of the features of the proposed change in the law. In one important respect the description is inadequate. There is one question to which no answer is given. Who will pay the bill for the Board of Registration in Chiropractic with its office, its officers, its clerks, and all the other machinery of adequate administration? The obvious reply is found in the proposed law: fees of the candidates applying for registration will be used to defray the expense of the Board, but where will they get the money? Of course it will not come from outside of Massachusetts but from the people of Massachusetts who think they already have enough bills to pay. In this time of financial stringency it has been pointed out on all sides that the unnecessary cost of government is one of our greatest burdens. It is especially ill-advised at this time to take any steps that will increase for the people of Massachusetts the cost of government. High pressure salesmanship has lost some of its charm. Massachusetts for thirty-eight years has found one Board for the registration of all practitioners of medicine sufficient for its needs. What change in the practice of

medicine has taken place in the past twenty-five or thirty-five years that makes another board anything but a waste of money from the point of view of governmental economy? No matter by what channel the money passes from the people to the treasury of the Commonwealth, it is the people of Massachusetts who will have to pay the bill. In accordance with the existing law, appropriations for the expense of carrying on the work of this new board (as of the boards already in existence in the Division of Registration) are actually to be made out of the general fund in the treasury of the Commonwealth and so will fall on every tax payer to the same extent whether he does or does not employ a chiropractor to treat himself or his family.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

LORD, FREDERICK T. A.B., M.D. Harvard University Medical School 1900. Visiting Physician, Massachusetts General Hospital. Clinical Professor of Medicine, Harvard University Medical School. President, American Association for Thoracic Surgery and the Massachusetts Tuberculosis League. His subject is "Pneumococcus Endocarditis." Page 767. Address: 305 Beacon Street, Boston.

PHIPPS, CADIS. A.B., M.D. Harvard University Medical School 1907. Director of the First and Third Medical Services, Boston City Hospital, and Physician-in-Chief of the Third Medical Service. Professor of Medicine, Tufts College Medical School. His subject is "Acute Bacterial Endocarditis." Page 768. Address: 587 Beacon Street, Boston.

MORRISON, HYMAN. B.A., M.D. Harvard University Medical School 1908. Associate Professor of Clinical Medicine. Tufts College Medical School. Visiting Physician, Beth Israel Hospital. His subject is "Subacute Streptococcus Endocarditis." Page 770. Address: 483 Beacon Street, Boston.

GARDNER, STUART N. A.B., M.D. Harvard University Medical School 1923. Assistant Physician, Salem Hospital. His subject is "Acute Monocytic Leukemia. A Case Report." Page 776. Address: 31 Chestnut Street, Salem.

STEWART, COLIN C. A.B., M.S., M.D. University of Pennsylvania 1926. His subject is "The Attenuation of Measles." Page 780. Address: Hanover, New Hampshire.

JOHNSTON, ALBERT C. B.S., M.D. Rush Medical College 1929. Pathologist to St. Louis Hospital, Berlin, New Hampshire. His subject is "Some Pathological Problems." Page 783. Address: Gorham, New Hampshire.

WHITE, JAMES C. A.B., M.D. Harvard University Medical School 1923. F.A.C.S. Instructor and Tutor in Surgery, Harvard University Medical School. Associate in Surgery, Massachusetts General Hospital. Consulting Surgeon, Massachusetts Eye and Ear Infirmary. His subject is "Progress in the Surgery of the Sympathetic Nervous System in 1931." Page 788. Address: Massachusetts General Hospital, Boston.

THE NOBEL PRIZE IN MEDICINE FOR 1932

THE news of the award of the Nobel Prize to Sir Charles Scott Sherrington of Oxford, jointly with Professor E. D. Adrian of Cambridge, as announced on October 27, will be received in this country with pleasure by all physicians. Professor Sherrington's work in London, Liverpool and Oxford on the physiology of the nervous system has brought him world-wide recognition. The earlier results of his researches were given in the form of the Silliman Memorial Lectures at Yale in 1906, later published as "The Integrative Action of the Nervous System" (1911), a book of primary importance. Since that date a long series of scientific papers have come from his laboratory at Oxford, where many American students have worked under his stimulating influence: Cushing, Forbes, Cobb and Viets of Boston; Weed of Baltimore; McCouch and Bazett of Philadelphia; Penfield of Montreal; and Fulton of New Haven, a list not by any means complete. Professor Sherrington also gave the Dunham Lectures at the Harvard Medical School in 1927, in which he reported the results of his subsequent researches. An important book from his laboratory, "Reflex Activity of the Spinal Cord" was reviewed in the *New England Journal of Medicine* of September 15, 1932.

Professor Adrian, whose particular field of investigation has been the mechanics of nerve conduction, recently lectured in Philadelphia on the Johnson Foundation.

By the work of these outstanding physiologists, our knowledge of the function of both the peripheral nerves and the spinal cord has been advanced. They have thrown new light on the special problems of reflex action, the integration of impulses and the nature of the nervous stimulation and its transmission. The Nobel Prize comes to them as a fitting recognition of their work, which has not only been fundamental in itself, but which has served to foster in their many pupils an investigative spirit in both physiology and neurosurgery, thus "keeping open the way to future discoveries".

IMPORTANT NOTICE

Chiropractic Referendum

The Massachusetts Medical Society, with the District Societies of Middlesex South, Norfolk and Suffolk, has arranged for a series of radio talks in opposition to the Chiropractic Referendum, Question No. 1 on the November ballot.

Date	*Time*	*Station*	*Speaker*
THURS., NOV. 3	6.25 P. M.	WNAC	DR. FRANCIS X. MAHONEY (Com. of Health of City of Boston)
FRI., NOV. 4	7.00 P. M.	WEEI	DR. SHIELDS WARREN
SAT., NOV. 5	6.25 P. M.	WNAC	DR. THOMAS J. O'BRIEN
MON., NOV. 7	7.00 P. M.	WEEI	DR. CHARLES E. MONGAN

Kindly invite your patients and friends to listen in.

MISCELLANY

FACTS ABOUT CHIROPRACTIC*

Submitted by E. S. Bagnall, M.D., Secretary and Treasurer of Essex North District Medical Society

In at least sixteen states unlicensed chiropractors exist, and of the total number of chiropractors in this country, nearly one-fourth practice without a license — i.e., illegally. Colorado, Iowa, Kansas, Montana, New Mexico, and South Dakota all have one chiropractor to every four physicians. In the United States as a whole, for every ten physicians there is one chiropractor. Contrary to the view held by some authorities, there is an even greater proportionate concentration of chiropractors than of physicians in large cities.

The growth of chiropractic has not been steady. From all accounts, the sect grew very rapidly from 1916 to 1923. In that year it seemed to have entered a depression which lasted until 1927 or 1928, followed by some numerical increase, but the good times of a decade ago have not returned, and chiropractic literature is full of statements to the effect that the handwriting is on the wall unless something radical is done. In 1930, a convention of chiropractors held in New York City was veritably a convention of wailers. Eyes were cast back fondly to the days when, as Mabel Palmer put it, "Chiropractors rode in Limousines with money jingling in every pocket." One Jeremiah gave chiropractic but a few years to live unless the sect revived the militancy of its youth. "Eight years ago," he said, "officials of the American Medical Association met in secret conclave in Chicago and adopted the slogan, 'Chiropractic must die.' They gave themselves ten years in which to exterminate it. . . . Are the medical doctors winning in their fight? Yes, gentlemen, they are. Are we being destroyed? Friends, we are."

In 1920, according to an investigation of the American Medical Association, there were seventy-nine chiropractic schools of which one, by far the largest, had over 2,000 students and was graduating new chiropractors at the rate of 1,500 a year. In 1927 there were only forty chiropractic schools, with a total attendance of not more than 2,000. At the present time there appear to be twenty-one active schools, with a probable attendance of 1,400. On the assumption that the gross incomes of chiropractors throughout the country average $4,200 the total amount spent annually for the services of these practitioners is approximately $63,000,000.

In many respects the history of chiropractic repeats that of osteopathy. Very early there arose the same quarrel between the "straights" and the "mixers," between those who believed in spinal manipulation as the one and only treatment and those who, having less faith in the omnipotence of chiropractic, reached out and embraced other therapeutic measures: diet, massage, osteopathy, colonic irrigation, physiotherapy, even drugs — in short, whatever the law allows or the chiropractor can use discreetly. Physiotherapy, especially, has been taken up by the chiropractors who show a fondness for large, benickled, mysterious-looking electrical treatment devices, lamps of various types, etc., all of which have their attraction for patients grown jaded of the chiropractic thrust.

Within the past two or three years the Palmer School has ceased to dominate chiropractic education, and in the same period more than half of the chiropractic schools, responding to a strong groundswell of opinion, have included physiotherapy in their curricula.

Gradually the chiropractors are edging their way into a position more in conformity with that of medicine. Thus we find the chairman of the Bureau of Research of the National Chiropractic Association writing:

"So long as we denied the existence of germs or denied the rôle of the germ in the specific processes of certain diseases, we made ourselves ridiculous and well deserved the epithet of ignoramus. When we acknowledged the facts of infection, but made it plain that we are in possession of a highly efficient mode of making it possible for the human body to fight bacteria invasion from within, we ceased to offend the intelligence of anybody and gained many converts to the chiropractic idea."

The following statement by a chiropractor at a United States Senate committee hearing is pertinent: "In fact, we had at one time 200 schools in the State of Michigan. They would start up with anything in a back parlor, for instance. We had a man over on East Capitol Street a few years ago who was advertising to teach chiropractic in 30 days for $10."

It has been stated that for many years 10 per cent of the revenues of the Palmer School went in-

*Much has been taken from the report of the Committee on the Costs of Medical Care.

to a fund used for securing chiropractic legislation in the various states. At the present time the chiropractors in the United States are organized into a National Chiropractic Association. This body is attempting to bring back the good times of olden days by an intensive publicity drive—a campaign known as the Life Line Program.

Two types of legal-aid membership are provided; one for chiropractors who hold a license to practice in their state, the other for chiropractors not so licensed. In either case, if members are arrested for practicing without a license or for going beyond the legally permitted scope of chiropractic or are involved in malpractice suits, they are defended by the legal staff of the association and are reimbursed for all fines and damages found. The legal-aid department is kept busy.

1. Part of their program consists in the circulation among laymen of a small chiropractic magazine for which the various chiropractors obtain subscriptions. In November, 1930, the secretary of the association reported that 100,000 subscriptions had been obtained, and new ones were coming in at the rate of 3,000 a week.

Beginning as an outlaw group practicing in defiance of the law, the chiropractors have been so successful in pushing their legislation that in all but six states they have won some measure of legal recognition. In thirty-two of these states, the chiropractic applicant for a license is examined by a separate board composed wholly of chiropractors. In Colorado, the medical board is obliged to give any and all applicants a chiropractic license without examination, if proof of graduation from a chiropractic school is shown. Six states — Delaware, Louisiana, Massachusetts, Mississippi, New York, and Texas—do not license chiropractors, as such, at all.

Where chiropractors have practiced illegally, it has been noticeably difficult to get juries to convict them. In over 80 per cent of the arrests of chiropractors, a president of one of the chiropractic associations has said, the juries have returned verdicts of not guilty. The secretary of the International Chiropractic Congress boasts that he has been arrested twenty-four times. In California, during one year, 450 chiropractors were arrested. They went to jail singing "Onward Christian Soldiers." Soon after, an amendment to the medical practice act legalizing chiropractic was put to a popular referendum and passed by a 145,000 majority.

Legal difficulties and the antagonism of the medical profession have not hindered the progress of the sect, but rather aided it. One chiropractic leader puts the case trenchantly:

"Let no chiropractor in our ranks think that we finally won legal recognition because a majority of the people were won over to our cause. We won because our minority was a militant minority. Those who were for us were for us all the way and back again. Our fight was their fight. They crowded the court rooms at our trial. They brought flowers and fruit to our jails. They carried our petitions and raised such an infernal howl that everybody in the community knew about us. And finally they got recognition for us. That was the fatal blow. Not because legal recognition was in itself a bad thing. Rather because it robbed us of the issue upon which our militant following had been built. Further because it robbed us of our own militant attitude toward organized medicine. . . .

"There are two points I would drive home with all the power at my command. The first is that we were most prosperous when we were a militant group fighting organized medicine. The second is that prosperity will return when we create an issue upon which we can again unite as a militant group fighting organized medicine."

He continues:

"What shall that issue be? . . . Probably the cleanest-cut issue just now is the issue of public hospitals or those supported by public subscription. These hospitals were not built by the medical profession. They were built by the layman. Money raised by taxation built them. They were not intended to create a monopoly in the healing profession. Christian Scientists helped to build them but can a Christian Scientist have his patients cared for there? Your patients helped to build them but can they have your services there? You know they cannot. We have the spectacle of states placing their stamp of approval on chiropractors yet refusing the facilities of state hospitals to them. Don't you think that is an issue upon which a militant public following can be built?"

How foolish it is for a community to maintain an elaborate machinery to prevent the spread of certain communicable diseases, and yet permit individuals to practice who are unable to differentiate diphtheria from pneumonia, or scarlet fever from measles. The matter is made worse by the fact that the chiropractors advertise themselves as, and are known as, doctors, and a large proportion of the population, unaware of the connotations of the letters M.D. and D.C., assumes that a doctor is a doctor, and that all are equally trained in the art of healing. This is even more true when a state licenses chiropractors, for the average layman is then apt to assume that the state vouches for their competency.

Chiropractic is rapidly changing. The rigid, "pure" dogma of the sect is becoming loose and broad, and softening into conformity with the ideas of medical science. The "straight" chiropractic treatment is losing prestige, and all sorts of auxiliary methods of treatment, some of them endorsed by medicine, are being adopted. The chiropractors are beginning to work for laws granting them a wider scope of practice. In short, the chiropractors now are like the osteopaths of fifteen or twenty years ago. Quite probably they will undergo a similar evolution. Their schools will become fewer in number but larger and better, and they will give a general medical course. In a word, we shall have half a dozen more inferior medical or near-medical colleges, and in the twilight of this sect a host of

poorly-qualified men become fused into the general body of medical practitioners. The prospect is not a happy one. It will have to be endured unless the life process of this sect can be aborted by bringing into play forces which will strike at the root of the sect and cause it to wither for lack of patrons.

PHYSICIANS REGISTERED BY EXAMINATION HELD IN JULY AND SEPTEMBER, 1932

COMMONWEALTH OF MASSACHUSETTS BOARD OF REGISTRATION IN MEDICINE

JULY EXAMINATION

Ainsworth, Leonard Bennett, 45 Sunset Avenue, Lawrence, Mass.

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Berman, Herbert Robert, 70 Bigelow Street, Quincy, Mass.

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Hunter, Thomas, Worcester City Hospital, Worcester, Mass.

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Kudish, Benedict, House of Providence Hospital, Holyoke, Mass.

Kvaraceus, Joseph Peter, 12 Intervale Street, Brockton, Mass.

Land, Herbert, 928 East 13th Street, Brooklyn, N. Y.

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Leddy, Andrew John, 275 Washington Street, Taunton, Mass.

Lundy, Joseph Augustine, Soldiers' Home Hospital, Chelsea, Mass.

Lyons, George Henry, 100 Ardale Street, Roslindale, Mass.

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Shipman, Thomas Leffingwell, Faulkner Hospital, Jamaica Plain, Mass.

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Steel, Selwyn Ladenberg, 25 Bennet Street, Boston, Mass.

Steele, Francis Joseph, St. Vincent Hospital, Worcester, Mass.

Thau, William, 72 Iffley Road, Roxbury, Mass.

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Troupin, Jack Leonard, Cambridge Hospital, 330 Mt. Auburn Street, Cambridge, Mass.

Vilker, Arthur H., 1510 North Shore Road, Revere, Mass.

Vohr, Fred Harold, 31 Park Avenue, Lee, Mass.

Webbe, Robert St. Clare Stanley, 600 King Street, Port Chester, N. Y.

Weiss, Carl Adolph, Lawrence General Hospital, Lawrence, Mass.

Welch, Robert Francis, 25 Smith Avenue, Brockton, Mass.

Wells, John Joseph, 61 Bonair Street, Somerville, Mass.

Willgoose, Dorothea May, Noyes Street, Needham, Mass.

ENDORSEMENTS OF NATIONAL BOARD REGISTRATIONS SINCE LAST REPORT

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Russell, Horace Pond, Massachusetts Memorial Hospitals, Boston, Mass.

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Serunian, Hoosic H., Pondville Hospital, Wrentham, Mass.

Silverman, Irving, 137 Shirley Avenue, Revere, Mass.

Sullivan, Charles Noyes, 50 William Street, Worcester, Mass.

Wallace, Jacob, 7 Wave Way Avenue, Winthrop, Mass.

Young, Alexander John, St. Luke's Hospital, Pittsfield, Mass.

MASSACHUSETTS BOARD OF REGISTRATION IN MEDICINE

Report of July 12, 13, 14, 1932 Examination

Medical School	Number Registered	Number Rejected
Boston University School of Medicine	11	1
College of Physicians and Surgeons, Boston	1	6
Harvard Medical School	11	2
Massachusetts College of Osteopathy	1	11
Middlesex College of Medicine and Surgery	10	21
Tufts College Medical School	28	5
Chicago College of Osteopathy	1	1
Cornell University Medical College	1	0
Dalhousie Faculty of Medicine	1	0
Des Moines Still College of Osteopathy	0	2
Georgetown University School of Medicine	1	1
Howard University School of Medicine	1	0
Johns Hopkins University School of Medicine	1	0
Kansas City University of Physicians and Surgeons	0	15
Kirksville College of Osteopathy	0	9
Laval University Faculty of Medicine	0	2
Long Island College Hospital	1	0
McGill University Faculty of Medicine	1	0
Missouri College Medicine and Surgery	0	1
Philadelphia College of Osteopathy	1	13
Rush Medical College	1	0
St. Louis College of Physicians and Surgeons	0	2
St. Louis University	0	1
University of Paris	0	1
University of Prague	1	0
University of Michigan	1	0
University of Montreal	0	1
University of Virginia	1	0

Western Reserve	1	0
Yale University Medical School	1	0
	77	95

PHYSICIANS REGISTERED BY EXAMINATION HELD SEPTEMBER 7, 1932

Barry, George Newton, Boston Dispensary, 25 Bennet Street, Boston, Mass.

Courville, Albert Leo, 8 Marlboro Street, Cornwall, Ont., Canada.

Huber, William McPherson, 1477 Beacon Street, Boston, Mass.

Walters, Charles Henry Austin, Belleville, Ontario, Canada.

Whiting, Charles Cosad, 118 East Main Street, Phelps, N. Y.

MASSACHUSETTS BOARD OF REGISTRATION IN MEDICINE

Report of September 7, 1932 Examination

Medical School	Number Registered	Number Rejected
Harvard Medical School	1	0
McGill University Faculty of Medicine	1	0
Queen's University College of Medicine	1	0
University of Pennsylvania	1	0
Washington University	1	0
	5	0

MR. JAMES F. BALLARD'S SERVICE AT THE BOSTON MEDICAL LIBRARY

On October 24, 1932, Mr. James F. Ballard, the Director of the Boston Medical Library, completed forty years of service in that institution. Mr. Ballard entered upon his life-work as a boy of fourteen in 1892 when the Library occupied a small building in Boylston Place. In 1900 a large new building was erected at 8 The Fenway where the Library now makes its home. Mr. Ballard held the title of Assistant Librarian for many years, receiving his title of Director in 1930.

Upon the occasion of his anniversary Mr. Ballard was informally remembered by his office workers and friends.

HARVARD MEDICAL SCHOOL APPOINTMENTS

ONE YEAR FROM SEPTEMBER 1, 1932

Hiram Houston Merritt, M.D., Instructor in Neuropathology.

Kenneth Clyde Farnsworth, M.D., Assistant in Anatomy.

Leo Rane, Ph.D., Assistant in Bacteriology.

David Davis, M.D., Assistant in Medicine.

Donald Storrs King, M.D., Assistant in Medicine.

Hyman Louis Kramer, M.D., Assistant in Medicine.

Wilfred Bloomberg, M.D., Assistant in Neurology.

Philip Solomon, M.D., Assistant in Neuropathology.

Hugo Linden Blair, M.D., Assistant in Ophthalmic Research (Howe Laboratory of Ophthalmology).

James Percy Baumberger, S.D., Research Associate in Medicine.

Eva Elizabeth Jones, Ph.D., Research Associate in Comparative Pathology.

Alwin Max Pappenheimer, Jr., Ph.D., Research Fellow in Bacteriology.

Samuel Hou Zia, M.D., Research Fellow in Bacteriology.

Harry Eagle, M.D., Research Fellow in Physical Chemistry.

John Fleming Brock, M.B., B.Ch., M.R.C.P., Research Fellow in Medicine.

Samuel Brown, M.D., Research Fellow in Medicine.

Frank Anthony Simon, M.D., Research Fellow in Medicine.

Mandel Ettelson Cohen, M.D., Research Fellow in Obstetrics.

Jacob Ellis Finesinger, M.D., Research Fellow in Psychiatry.

Joseph Jules Michaels, M.D., Research Fellow in Psychiatry.

William Frederick Roth, M.D., Research Fellow in Psychiatry.

Conrad Wall, M.D., Research Fellow in Psychiatry.

Samuel Jacob Beck, Ph.D., Research Fellow in Psychology.

Harold Francis Robertson, M.B., Research Fellow in Surgery.

Appointment: From September 1, 1932 to March 1, 1933:

Knox Henderson Finley, M.D., Research Fellow in Neuropathlogy.

APPOINTMENT OF DR. HENRY BAKER

Dr. Henry Baker, 483 Beacon Street, Boston, has recently been appointed Assistant Professor of Medicine, Tufts College Medical School. Dr. Baker is a graduate of Harvard College and Harvard Medical School, a member of the staff of the Boston City Hospital and the Beth Israel Hospital and a Councilor of the Massachusetts Medical Society, Norfolk District.

THE BOSTON BETTER BUSINESS BUREAU AND THE CHIROPRACTIC REFERENDUM

The *Fitchburg Sentinel* has recently referred to the action of the Boston Better Business Bureau in regard to the Chiropractic Referendum (No. 1) which will appear on the November ballot. This Bureau has advised a "No" vote on the question, and the *Sentinel* states, "It (The Boston Better Business Bureau) is non-political, non-sectarian and so far as we have ever heard untrammeled in every or any way. One certain thing is that it is not controlled by the medical profession. We have followed its work, and the work of the National Better Business Bureau, for years, and have found it most uniformly sound in its conclusions." To quote the *Sentinel* further:

"The *Bulletin* concludes with these words: 'From the above it seems clear that the passage of this law might result in the registration of hundreds of people, legally licensed to cure the sick and diseased, but having no education, experience, or training in this field. The Better Business Bureau recently solicited the opinion of the boards of health throughout the state as to whether the passage of this bill was in the interests or contrary to the interests of the public. The replies received from the boards of health are almost unanimously to the effect that the proposed law is inimical to public health.'"

CORRESPONDENCE

ACTIVITIES RESPECTING THE CHIROPRACTIC INITIATIVE PETITION

The Massachusetts Medical Society
Committee Room
8 The Fenway, Boston, Mass.

Editor, The *New England Journal of Medicine,*

The following is roughly what has been done to date concerning the Chiropractic Initiative Petition:

When the Committee on Legislation was confronted with this political problem, it sought the advice of the ablest men in both parties. They patiently considered the subject and advised us that unless we influenced the physicians and members of their families to make personal contact with as many voters as possible, we would be defeated. They pointed out the strategic position which we had with physicians in every hamlet, town and precinct. They agreed that radio, newspaper and advertising by mail were less valuable than the personal contact method. They advised the use of circulars if distributed by the physician personally. Being novices we followed their advice.

We have addressed the members of the Council and have circularized the officers of the district societies, the Councilors, the 280 members of the Auxiliary Committee, the superintendents of all hospitals, the chiefs of all staffs, the Boards of Health and most welfare and public health organizations.

We have interested the officers of the Massachusetts State Pharmaceutical Association and at their own expense they have mailed circulars to their more than twenty-two hundred members. The pharmacists have been instructed to place posters in their stores and speak to their customers.

The Massachusetts State Nurses' Association has placed a column in its Bulletin and permitted one of our members to address the Association at its Annual Meeting in Boston. It numbers about five thousand and the members are most active.

The President of the Massachusetts Dental Society has been interviewed and his organization is interested in this educational campaign.

The President of the Bar Association and many kindred groups have had our problem outlined to them.

The Massachusetts Tuberculosis League has released its Executive Secretary from ordinary duties that he may devote his entire time to organizing a lay committee to oppose this bill. They, too, have had a circular printed at their expense which is being widely distributed.

We have interested the officers of the Massachusetts Osteopathic Society and they have many workers distributing circulars at their own expense.

We have had an editorial printed in the *Gazetta del Massachusetts* in opposition to the chiropractic bill and, through the President of the Massachusetts Italian Medical Society, copies of this paper are being distributed throughout the State without expense to our Society.

Through one of the students at the Tufts College Medical School, we have been able to reach the voters of Greek extraction.

We have influenced the Boards of Health of some places and circulars have been distributed in those particular cities and towns at their expense. We have written personal letters to the Boards of Health throughout the State in order that they may influence the editors of their local papers to write editorials or first-page notices that the general public may be properly instructed concerning the danger of permitting chiropractors to handle communicable diseases.

The United Drug Company has notified the manager of each of its two hundred and thirty-five stores to vigorously oppose this bill.

Members of the Legislative Committee and well-informed members of the Society have been giving addresses almost nightly from New Bedford to Pittsfield.

In many cities much has been done. In Worcester many thousands of circulars have been distributed at the expense of the members of that district. Their legislative committee has instructed certain laymen who in turn have lectured before many groups and their local newspapers have been most generous with editorials on the subject. The physicians of many cities have raised funds ranging from $50 to $350 which money is being expended judiciously by committees.

The students at Harvard, Boston University and Tufts Medical School and those at the Massachusetts College of Pharmacy have been asked to coöperate in obtaining votes in opposition to the chiropractic bill. As these schools average about five hundred students each, we expect much aid from them.

Certain physicians, at their own expense, are to pay workers who will instruct voters on the day of the election.

The Society is limited as to expenditures but we feel that public-spirited physicians might well contribute unlimited amounts for the issuing of postal cards and, what would be more important, for a quarter-page advertisement in the daily newspapers a few days before the election. We heartily recommend this last suggestion and individual members of the Legislative Committee will contribute and

respectfully solicit contributions from physicians and their patients for this important method of instructing the general public.

Sincerely yours,

COMMITTEE ON STATE AND NATIONAL LEGISLATION.

RECENT DEATH

GILBERT—DR. MILTON JOHN GILBERT died at the Fall River Hospital, October 26, 1932. He was the son of Dr. John Gilbert and had been medical inspector of the public schools for fifteen years.

He graduated from the College of Physicians and Surgeons in Boston in 1906 and joined the Massachusetts Medical Society in 1925. He is survived by his parents, his widow and three sons.

OBITUARY

RESOLUTIONS ADOPTED BY THE BOSTON CITY HOSPITAL ON THE DEATH OF DR. ABRAHAM S. TROUPIN

The sudden unexpected death of Abraham S. Troupin has removed from the medical community a man who had endeared himself to his associates in an important manner over a period of years, only too brief in number. During this time, however, there was revealed a usefulness to his patients, a great capacity of service, and a skill in his chosen department of medicine which won high commendation from his fellows.

Appointed as Assistant to the Visiting Surgeons in Gynecology and Obstetrics at the Boston City Hospital in 1927, he was soon promoted to Junior Visiting Surgeon, and in 1931 to the rank of Assistant Visiting Surgeon. His teaching affiliations were those of Assistant in Gynecology at the Harvard Medical School, and only recently he had been appointed Assistant Professor of Obstetrics at Tufts College Medical School. He was a member of the Massachusetts Medical Society, the New England Obstetrical and Gynecological Society and the American Medical Association.

The Staff of the Boston City Hospital shares with the associates of Abraham S. Troupin a sense of loss of service to the hospital, and a deep sorrow for his family who most closely feel the shock of his death.

Resolved: that this memento be inscribed in the minutes of the Senior Staff.

P. F. BUTLER, *Secretary Senior Staff.*

October 27, 1932.

NOTICES

AN ANONYMOUS CONTRIBUTION

This *Journal* is in receipt of a complaint in which some sort of a financial agency is characterized as a "Racket" operating at the expense of physicians and requesting that doctors should be warned about this concern.

The writer omits to sign his name and thus places himself outside the pale of good standing.

We are interested in serving the medical profession but until specific charges are made and a signature attached, letters cannot be given serious attention.

REMOVAL

WILLIAM D. MCFEE, M.D., announces his office removal to 31 Bay State Road, Boston.

CORRECTION

In the *Journal* of October 20 on page 716 in "This Week's Issue" under the name of Francis D. Donoghue the words "Geneva 1931" should have followed Chairman of U. S. Delegation, Congress Industrial Accidents and Occupational Diseases.

CLINICS IN MEDICINE AT PETER BENT BRIGHAM HOSPITAL

On Thursdays at 3:30 P. M. in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard, and Physician-in-Chief, Peter Bent Brigham Hospital, will give a series of clinics which members of the medical profession are cordially invited to attend. The next clinic will be given on November 10.

Also physicians and medical students are invited to attend ward rounds of the Medical Staff of the Peter Bent Brigham Hospital held from 10-12 o'clock on Saturdays, as well as a clinical-pathological demonstration immediately following these rounds.

LECTURES AT THE HARVARD MEDICAL SCHOOL

The George W. Gay Lectures on "Medical Ethics"—Amphitheater C at 5 P. M. Tuesday, November 8, Thursday, November 10—Dr. Frederic J. Cotton of Boston.

Lecture on "The Care of the Patient" — Amphitheater C at 5 P. M. Tuesday, November 15 — Dr. Roger I. Lee of Boston.

RADIO HEALTH MESSAGES

NOVEMBER-DECEMBER, 1932

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZ. Tuesdays, 4:15 P.M.

November

8 Our Vitamins
15 The Health Examination
22 Only a Sore Throat
29 Anemia or the Lack of Blood

December

6 Sickness and Character
13 How Are You?
20 Chorea or St. Vitus' Dance
27 Hemorrhoids

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 12:30 P.M.

Health Subjects of Public Interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 4:50 P.M.

Questions on Health and Prevention of Disease may be sent to: Radio Health Forum, State Department of Public Health, State House, Boston.

REPORTS AND NOTICES OF MEETINGS

WACHUSETT MEDICAL IMPROVEMENT SOCIETY

The regular meeting of the Wachusett Medical Improvement Society was held on Wednesday, November 2, at the Rutland State Sanatorium.

The speaker was Dr. Benedict Fenwick Boland of Boston who offered a combined gynecological and obstetrical program, illustrated by lantern slides. The first part was entitled "Conization of the Cervix Uteri." The second part was on the "Separation of the Symphysis Pubis during Delivery."

ROBERT J. NORTHRIDGE, M.D., *Secretary.*

HAMPDEN DISTRICT MEDICAL SOCIETY

The fall meeting of the Hampden District Medical Society was held October 18, 1932, at the Monson State Hospital at the invitation of Dr. Morgan B. Hodskins. A scientific program followed an excellent luncheon. Papers and case histories were presented by Drs. R. H. Guthrie, P. I. Yakovlev and Calvert Stein.

The program indicated that the hospital was interested, not only in the care of epileptics but in research as well.

In the business meeting the "Chiropractic Bill" was discussed. Methods of informing the public were considered.

Dr. John M. Birnie asked for suggestions concerning the program of the State Society for Post Graduate Study. Many useful plans were suggested.

Dr. Fred H. Allen, the president, presided. Dr. H. L. Smith is Secretary-Treasurer.

J. A. WHITNEY, M.D., *Reporter.*

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

The quarterly meeting of the Worcester North District Medical Society was held at the Gardner State Colony on October 25. Dr. A. Warren Stearns, Commissioner of the Department of Correction and Dean of Tufts College Medical School, addressed the members, and Judges of the Courts in the District were invited to hear him. Dr. Robert A. Rice, president, presided.

Dr. Stearns said in part, "Between eighty and ninety per cent of the boys at Concord Reformatory come from broken homes. . . . Throughout the ages efforts to prevent crime, especially among boys, by physical punishment have failed. There are three problems which must be faced—the boy who is ill. the boy who is poor and the boy who is bad. The general attitude toward the bad boy has been retaliation. The situation must be solved by study and corrective treatment. Knowledge of personal qualities and social positions of the offenders are essential to properly handle their cases."

Prisons, Dr. Stearns characterized as necessary evils but never a cure for crime. He complimented district court judges and probation officers for their humane consideration of juveniles.

Dr. E. A. Hunter of the state colony explained a plan by which delinquent children, summoned into court, are studied. In Fitchburg, he said, in half of sixteen cases it was found that parents were at fault because they made excuses for their children.

About sixty members of the Worcester North District Medical Society attended the meeting and enjoyed Dr. Stearns' interesting address.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Ninety-First quarterly meeting of Essex North District Medical Society was held at Raymond's Sunnyridge Farm in Amesbury, on October 19, 1932, at 1 P. M., in conjunction with the Essex South District. There were eighty-nine present.

After an excellent shore dinner Dr. Otis P. Mudge of Amesbury, president of Essex North, introduced Dr. H. G. Stetson, president of the Massachusetts Medical Society, who discoursed on problems confronting the society at present. He stressed the importance of due care on the part of the censors before recommending admission of candidates to the society. He discussed briefly the chiropractic referendum recommending the "No" vote and stressed the work of the Public Relations Committee in improving the understanding between public health organizations and the practitioner. He mentioned that the next project of the committee was the ironing out of some of the difficulties in the workings of the Industrial Accident Law. He mentioned the projected Post-Graduate Instruction which is in preparation by a special committee and said that the idea was not to make specialists, but to make it easier for the general practitioner to keep in step with the march of progress.

Dr. McFee, of Haverhill, for this committee, asked that the physicians help the committee in formulating the most desirable plans and methods for the carrying of this postgraduate instruction to the doctor. The secretary stated that he would send

copies of the various plans to the hospitals in the district and ask for preferences to be indicated by each member of the respective staffs.

The president then introduced Dr. Frank R. Ober, Assistant Dean, Courses for Graduates, Harvard Medical School, who discussed, with refreshing perspective, the problem of arthritis. Dr. Ober said that of the 145,000 reported arthritics in Massachusetts, many were receiving inadequate treatment. This makes an average of 22 arthritics for each physician. He mentioned the importance of eliminating focal infections, including teeth, tonsils, sinuses, intestinal and urinary tracts, but urged that these were only a part of the problem. He discussed the importance of allowing plenty of time for the food to be properly assimilated in the intestinal tract, indicating the bad results sometimes arising from cathartics. Dr. Ober mentioned that dram doses of barium are an excellent indicator of traffic rates in the intestinal tract, seventy-two hours being a fair time for the elimination of the barium after administration and urged that stool examination be not neglected as sometimes the most important indications for treatment were based on the findings of an acid stool with starch indigestion. He felt that the thyroid was not a primary cause but might well be contributory. Summing up the etiology he expressed the opinion that self-indulgence was the commonest and most important factor as regards eating, emotional instability, and too much or too little exercise. He pointed out that observations at the Children's Hospital in Boston showed that children developed an acetonuria as the result of homesickness or fear.

In discussing pathology he expressed preference for the old classification of Richardson and Nichols. In proliferative arthritis one gets granulation tissue between the synovial membrane and cartilage, next a pannus in the joint causing the ankylosis with atrophy of the cartilage. The x-ray shows fewer spurs in this, the type of early life. Secondly, the degenerative type with eburnation of the ends of the bone with spur formation causes locking of the joints.

Experimental work, which showed that bacterial, chemical or traumatic influences all had similar effects on the joints in the way of producing arthritis, was cited.

Dr. Ober urged the importance of early diagnosis, the cervical spine usually being affected first. He stated that a lateral x-ray often demonstrates the disease before the symptoms are well-established, and a practical office test is the presence of a limited lateral motion in the cervical spine. These early cases are often associated with aching at the base of the neck, stiffness on getting out of bed in the morning and spasm of the skull trapezius.

In treatment he quoted the old saying, "It is more important to know what sort of patient had the disease than to know what disease the patient had." He stressed the value of Vitamin B, mentioning Harris Concentrated B tablet, Bemax and Magic Yeast, as good forms, the Fleischmann Yeast being about 80% starch. He emphasized the importance in most cases of cutting down the starches and sugars and in the matter of maintenance of muscular tone to lessen joint stress. He urged the education of the patient in the manner of setting and relaxing the muscles about the joints. In the proliferative type he urged that while splinting and bed rest were desirable for acute cases, motion should be used a few times a day within the limits of pain to prevent adhesions.

In the discussion which followed he said that 85% of the cases that coöperate intelligently should be relieved of their symptoms, leaving only the joint destruction which had already occurred when treatment was initiated. He said the diathermy machine in his office was used about once in six months.

The talk created much interest and informal discussion was continued for a long time after the meeting adjourned about 5 P. M.

PLYMOUTH DISTRICT MEDICAL SOCIETY

The October meeting of the Plymouth District Medical Society was held in Grand Army Hall, Abington, Mass., on October 20, 1932. The meeting was called to order by the president, Dr. Charles G. Miles. The minutes of the mid-summer meeting were read by Dr. George A. Moore, secretary. Dr. Moore reported that he had received literature concerning the chiropractic bill that is to be voted on in the November election. He had this literature for distribution and it was freely circulated among the members.

Dr. Thomas H. McCarthy, past president of the society, talked on the chiropractic bill. Dr. McCarthy said that in his years of membership in the Massachusetts Medical Society, never before had there been such a menace to the general practitioner as is now before them. The income of the general practitioner is at a very low ebb. Our people, in distressing circumstances, destitute in a great many cases and out of work, continually seeking employment, with sickness upon them, need doctors to administer to their ills.

Dr. McCarthy said, "If this bill passes it will add materially to the millions of dollars annually taken from the practitioners of medicine. I am appealing to you to get busy as you never have before and contact your neighbors and friends. Tell them to vote NO on this proposition."

He reported the action taken by the Council at its last meeting and told the Society that he proposed writing personal letters.

"Each member should write 25 letters to his friends telling them, as a personal favor to him, to vote 'NO' on this proposition." He said, "I am placing it before the Plymouth District Medical Society for action as an organized body, because we want to go on record showing that the Plymouth District Medical Society is 100% loyal to the medical profession of Massachusetts. No doubt there are

many physicians and surgeons throughout the State of Massachusetts who, when they understand how it will affect their incomes, will loyally stand by a proposition of this kind. Unfortunately there are many in our society who lag and are willing that the other fellow do the work and they, like drones in a beehive, live at some one else's expense doing nothing not only for their own benefit but for that of thousands of other physicians in the State. No words of mine can express what it will mean to you if this bill passes. If the general practitioner has to compete with a large influx of chiropractors throughout the State of Massachusetts, he will find that his income will be even lower than now. I hope and pray that the members of the Massachusetts Medical Society will see the justice of my stand for their welfare."

Dr. McCarthy's proposition was unanimously adopted by the Society and a committee consisting of Dr. Thomas H. McCarthy, Dr. George A. Moore, and Dr. Peirce H. Leavitt was appointed by President Miles to take immediate action.

Dr. John J. Condrick, Chairman of the Committee on Clinics, gave a detailed report for his committee. Among other things, Dr. Condrick said he felt that the Plymouth District Medical Society had for years done everything it could to uphold the high standard of the nose and throat specialists. Dr. Condrick said that the nose and throat specialists were willing to take out tonsils for $5.00, and $8.00 for people who were in a position to pay the regular fee. He said further that if these operators, so-called specialists, were willing to do this work for practically nothing, he felt the fact that they wished to lower their charges to such a ridiculously low degree was no concern of the Society. It is an entirely different proposition for people who are poor and unable to pay. Nobody objects if the physicians and surgeons wish to give their services to the worthy poor. Dr. Condrick said that he for one was tired of trying to help those not willing to help themselves. The report was accepted as progressive.

The speaker of the morning, Dr. Abraham Myerson, Professor of Psychiatry at Tufts College Medical School, was then introduced. Dr. Myerson took for his subject "The Cerebrospinal Fluid Syndrome." He went into detail in regard to the latest practical methods of diagnosis in neurology and mental diseases and demonstrated that he was master of the situation. He gave a very instructive and interesting discussion of his subject. A rising vote of thanks followed his excellent address.

After the meeting the Women's Relief Corps served a most excellent dinner in the dining hall. Between 50 and 75 physicians from Plymouth, Bristol and Norfolk Counties were present.

The next meeting will be held at the Moore Hospital, Brockton, on November 17, 1932.

THE NEW ENGLAND ROENTGEN RAY SOCIETY

The New England Roentgen Ray Society held its first meeting of the year on Friday evening, October 21, in the Thorndike Amphitheatre at the Boston City Hospital. Dr. Blackett presided, and after calling the meeting to order introduced Dr. P. F. Butler as the chairman for the evening.

Dr. D. O. McKee presented the case history and x-ray films of a 14 year old boy with adenocarcinoma of the descending colon. There was a history of pyuria and change of bowel habits. The chief symptoms were referable to a secondary anemia, and consisted of weakness and easy fatigability. There was a mass in the left upper quadrant thought to be the spleen, and a separate tumor mass palpable just above the iliac crest. There was gross blood in the stools. X-ray films showed narrowing and irregularity of the descending colon.

Laparotomy revealed a carcinoma of the colon, a normal kidney and enlarged spleen. The enlargement of the spleen was probably due to the proximity of this organ to the inflammatory condition in the carcinoma. The pyuria was explained on a similar basis as an extension of the inflammation to the ureter. The case was of especial interest in that this type of malignancy is quite rare in so young a person.

The second case was that of a congenital malposition of the colon and appendix. The symptoms caused by the subacute inflammation of the appendix were referable to the epigastrium. The x-ray revealed the position of the cecum in the left upper quadrant and was of great value to the surgeon, who had first intended to make his incision in the right lower quadrant.

Dr. M. I. Smedal discussed the condition of vertebral epiphysitis. This condition gives rise quite frequently to adolescent kyphosis. In spines that have a hereditary tendency, and in those subject to unusual mechanical strains, the nucleus pulposus expands unduly and as a result we find a narrowing of the vertebral bodies anteriorly. When this narrowing take place anteriorly in successive bodies it gives rise to a marked kyphosis.

Forty per cent of the spines examined at autopsy show some such changes. Most of these are without symptoms. The x-ray findings are those of a widened intervertebral space posteriorly due to nuclear expansion together with irregularity of the superior and inferior borders of the vertebrae. The symptoms noted are chiefly pain and backache. The diagnosis is made on the basis of a deformity and the x-ray findings.

Dr. Norton in discussing the treatment of these cases emphasized that more than posture clinics are necessary in many cases and that scientific appliances are often required to effect a permanent cure.

Dr. P. E. Tjvnan discussed his studies of joint changes in the pelvic girdle. There are three conditions that bring about abnormal mobility in these structures. They are those of pregnancy, men-

struation and trauma. Studies have been made of the changes during pregnancy. The normal range of motion between the bones of the symphysis is 1.5 millimeters. During pregnancy this is increased to six mm. This reaches its maximum at the time of parturition, and it returns to normal six weeks after delivery.

Frequently women will complain of low back pain, difficult walking and tenderness over the symphysis after delivery. These symptoms may be due entirely to continued mobility of the pelvic joints. The Gynecological Service has recently devised a belt, the purpose of which is to pull the bones of the symphysis pubis together. This gives relief to these patients, and hastens the return to normal of the joint. The increased mobility occurring in this joint during menstruation is being studied at present by Dr. Tivnan.

Dr. A. M. Moloney discussed the x-ray diagnosis of conditions about the knee joint. One case was that of syphilitic endarteritis giving a localized eburnation of the upper end of the tibia. The edges of the shaft showed haziness and irregularity of outline suggesting osteogenic sarcoma. Another case gave a history of injury to the knee. X-ray films showed new growth of bone with some bone destruction in the shaft. The surgeon was convinced that the lesion was osteogenic sarcoma, but biopsy revealed a benign exostosis.

Dr. F. W. O'Brien described an interesting case of sclerosing osteomyelitis (Garrè's disease), which was first diagnosed as Ewing's sarcoma. The patient gave a history of a former acute osteomyelitis and repeated injuries to the knee joint. There was a spontaneous fracture at the onset of the present illness and a pathological diagnosis of Ewing's sarcoma was made. Three series of x-ray treatments were given and no change took place in the lesion. On the basis of this the pathologist was asked to reconsider his diagnosis. This time the diagnosis of sclerosing osteomyelitis was made, which has since been confirmed by Dr. Ewing.

These diseases often give similar pictures. Both usually begin with fever, leucocytosis and night pain of a deep, aching character. The Ewing tumor responds to x-ray while the sclerosing osteomyelitis does not. Usually the latter has an antecedent history of acute osteomyelitis of a series of infections. The prognosis is favorable for life.

Dr. Max Ritvo showed films from several interesting cases. One was that of a branchial cleft fistula opening in the neck. Lipiodol injection demonstrated a communication with the bronchus at about the level of the bifurcation of the trachea.

Several slides were shown by Dr. Ritvo illustrating the x-ray studies made of breasts. In normal the canaliculi and tubules can be seen merging toward the nipple. If a mass grows into the tubules it is usually malignant; while if it compresses them it is usually benign. X-ray will often disclose a gland in the axilla when it is not palpable.

Dr. Butler concluded the program with a discussion of Beck's paste in the visualization of cavities and sinuses. This paste is now available in tube form and is easily introduced into the sinus or cavity. After it has been introduced, it is well to have the patient walk about for a short time in order that the paste may work itself into all parts of the tract.

BOSTON ORTHOPAEDIC CLUB

PROGRAM

Symposium on Arthritis

I

November 14, 8 P. M. John Ware Hall, Boston Medical Library.

1. Nature of the Malady, Incidence and Economic Importance—Dr. Robert B. Osgood.
2. Classification and Pathologic Changes — Dr. Chester Keefer (of the Thorndike Memorial Laboratory, Boston City Hospital).
3. The Infection Hypothesis—Dr. Ralph H. Boots (of the Department of Medicine, College of Physicians and Surgeons, Columbia University, and the Arthritis Clinic, Presbyterian Hospital, New York City).
4. Differentiation in Diagnosis and Etiology of Rheumatoid Arthritis—Dr. Walter Bauer (of the Robert W. Lovett Memorial for the Study of Crippling Disease, Harvard Medical School).

II

December 12, 5 to 6:45 P. M. Robert Breck Brigham Hospital, Parker Hill.

Clinic—illustrating operative and non-operative orthopaedic treatment.

III

December 12, 8:15 P. M. Harvard Medical School, Amphitheatre, Building B.

1. General Aspects of Treatment—Dr. George R. Minot.
2. The Prevention of Deformity — Dr. Loring T. Swaim.
3. Surgical Aspects of Treatment — Dr. Philip D. Wilson.
4. Prevention and Community Organization — Dr. Frank R. Ober.

All members of the profession are cordially invited to attend.

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the office of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, November 15, 1932, at 12 noon.

The speaker will be Henry A. Christian, M.D., Physician-in-Chief, Peter Bent Brigham Hospital, and his subject, "Bedside Diagnosis of Heart Disease."

The usual luncheon will be served at 1 o'clock. All physicians are cordially invited to attend. Come and bring guests who are in the medical profession.

NEW ENGLAND HEART ASSOCIATION

CLINICAL COURSE

The New England Heart Association is planning to give a limited number of lectures similar to those held during the past two winters. The first meeting will be at the Boston Medical Library, Wednesday afternoon, December 7, at four-thirty P. M.

The subject will be Acute and Chronic Pericarditis.

1. Medical Aspects. Dr. S. A. Levine.
2. Pathological Aspects. Dr. Charles F. Branch.
3. Surgical Aspects. Dr. E. D. Churchill.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The Middlesex East District Medical Society will hold its next meeting on Wednesday, November 9, at the Calumet Club, Winchester.

Dr. Chester M. Jones, physician to the Massachusetts General Hospital, Boston, will discuss "The Sick Liver."

Luncheon will be served at 1:00 P. M.

ALLAN R. CUNNINGHAM, M.D., *Secretary.*

WORCESTER DISTRICT MEDICAL SOCIETY

HAHNEMANN HOSPITAL, WORCESTER—WEDNESDAY, NOVEMBER, 9, 1932

5-6:30 P. M. Moving Pictures.

1. Immediate Repair of Perineum after Delivery.
2. Transurethral Prostatic Resection.
3. Activities at Hahnemann Hospital.
4. Carcinoma of Penis (Radical Operation).

6:30 P. M. Dinner.

7:30 P. M. Business Meeting and Scientific Program.

1. Pyloroplasty. Paper by Dr. Edgar A. Fisher. (10 minutes.) Discussion by Dr. Michael Fallon.
2. Report on a Series of Caesarean Sections. Paper by Dr. Joel Melick. (10 minutes.) Discussion by Dr. William E. Denning.
3. Prostatism. Paper by Dr. Lester Felton. (10 minutes.) Discussion by Dr. Bancroft Wheeler.
4. Acute Aleukemic Leukemia. Paper reporting a case by Dr. Gerald Shelby and Dr. William Freeman.

NOTICE

We must all continue our work toward defeating Referendum Number One. Remember that Election Day is only a few days away. Are you doing your part?

EDWARD R. LEIB, M.D., *President,*
ERWIN C. MILLER, M.D., *Secretary.*

SOCIETY MEETINGS
CONGRESSES AND CONFERENCES

November 4—The William Harvey Society. See page 764, issue of October 27.

November 8 and 10—The George W. Gay Lectures on "Medical Ethics." See page 808.

November 10—Clinics in Medicine at Peter Bent Brigham Hospital. See page 808.

November 14 and December 12—Boston Orthopaedic Club. See page 812.

November 15—Lecture on "The Care of the Patient." See page 808.

November 15—South End Medical Club. See page 812.

November 21—Boston Medical Library. Medical History Club. See page 723, issue of October 20; also see page 765, issue of October 27.

December 7—American Board of Obstetrics and Gynecology. Announcement of Coming Examinations. See notice on page 557, issue of September 22.

December 7—New England Heart Association. See notice elsewhere on this page.

February 6-10—The American College of Physicians. See page 765, issue of October 27.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

January 4—Wednesday, Semi-Annual Meeting. Program to be announced.

May 3—Wednesday, Annual Meeting. Program to be announced.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Tentative Program
1932-1933

Thursday, November 3, 1932—Censors' meeting at Salem Hospital, 3:30 P. M.

Wednesday, November 9, 1932—Salem Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Charles F. Branch, Boston.

Wednesday, December 7, 1932—Beverly Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Robert Greenough. Subject to be announced later.

Wednesday, January 4, 1933—Danvers State Hospital, Hathorne. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. George Kline. Subject to be announced later.

Wednesday, February 1, 1933—Council Meeting, Boston.

Wednesday, March 8, 1933—Lynn Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Soma Weiss, Boston City Hospital. Subject: Hypertension.

Wednesday, April 5, 1933—Essex Sanatorium, Middleton. Clinic 5 P. M. Dinner 7 P. M. Speaker and subject to be announced later.

Thursday, May 4, 1933—Censors' Meeting, Salem Hospital, 3:30 P. M.

Tuesday, May 9, 1933—Annual Meeting. Salem Country Club, Forest Street, Peabody. Golf in afternoon; green fee $2.00. Dinner at 7 P. M. Speaker: Dr. Burgess Magrath. Subject to be announced later. Ladies invited.

R. E. STONE, M.D., Secretary.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

The program of the Middlesex East District Medical Society for the coming year is as follows:

November 9—See notice elsewhere on this page.

January 11 at Wakefield.

March 15 at Melrose.

May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, Secretary.

NORFOLK DISTRICT MEDICAL SOCIETY

November 29—Norwood Hospital, Norwood, Mass. Time to be announced. Papers by the Norwood Hospital Staff.

January 31, 1933—Roxbury Masonic Temple, 8 P. M. Subject: "Stomach Surgery"—Motion pictures. Dr. William R. Morrison.

February 28, 1933—Time and place to be announced. "The Diagnosis and Treatment of Anemia," by Dr. William P. Murphy.

March 28, 1933—Time, place and subject to be announced. Speaker will be Dr. J. P. O'Hare.

May—Annual Meeting. Time, place and subject to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

November 17—At the Moore Hospital, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

November 3—The Censors of the Suffolk District Medical Society will meet for the examination of candidates

at the Medical Library, No. 8 The Fenway, Thursday, November 3, 1932, at 4 o'clock.

LELAND S. McKITTRICK, M.D., Secretary.
205 Beacon Street, Boston.

Wednesdays at 8:15 P. M.

November 30, 1932—Clinical Program, Peter Bent Brigham Hospital.

January 25, 1933—General Meeting in association with the Boston Medical Library at the Boston Medical Library. Titles and speakers to be announced later.

March 29, 1933—Clinical Program, Massachusetts General Hospital.

April 26, 1933—Annual Meeting. Election of Officers. Scientific Program—Titles and speakers to be announced later.

The Medical Profession is cordially invited to attend all of these meetings.

WILLIAM H. ROBEY, M.D., President,
LELAND S. McKITTRICK, M.D., Secretary,
HILBERT F. DAY, M.D., Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

Schedule of meetings for 1932-1933:

Thursday, November 3—Fall Censors' meeting. See page 636, issue of October 6.

Wednesday, November 9—Dinner and scientific meeting at the Worcester Hahnemann Hospital. See page 813.

Wednesday, December 14—Meeting at the Worcester Chamber of Commerce Auditorium. Paper by Dr. Joseph Meigs of Boston, entitled "Uterine Bleeding."

Wednesday, January 11—Dinner and meeting at the Worcester City Hospital. Paper by Dr. Morris Fishbein, Editor of the Journal of the American Medical Association.

Wednesday, February 8—Dinner and scientific meeting at the Worcester State Hospital. Program to be announced later.

Wednesday, March 8—Dinner and scientific program at the Memorial Hospital, Worcester. Program to be announced later.

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, Secretary.

BOOK REVIEWS

Epidemic Encephalitis: Etiology, Epidemiology, Treatment. Second Report by the Matheson Commission. New York: Columbia University Press, 1932. 155 pages.

The Matheson Commission of New York issued its first report on epidemic encephalitis in 1929 (*New Eng. J. Med.* 201:1213, Dec. 12, 1929). This contained a study of the literature on the subject through 1928. The present volume, which is the second report, reviews the literature up to the end of 1929. Since the summer of 1929 the Commission has been located at the Neurological Institute, New York, working with a group of patients suffering from acute chronic epidemic encephalitis and trying out various methods of treatment; more than 500 patients have been studied. The clinical reports dealing with this large group are to be published later.

The Commission finds at the present time that the etiology of epidemic encephalitis is still not known, in spite of the work of many investigators. An investigation of a similar disease, postvaccinal encephalitis, is also included in this report, which indicates that the etiology of this malady also remains unsolved at the present time.

The most important part of the volume is concerned with the treatment of epidemic encephalitis. The Commission feels that the arsenicals are to be recommended in the convalescent or in the chronic stage of epidemic encephalitis for their tonic effect. Atropine is also of value in the treatment of the symptoms in the chronic stage. The efficiency of convalescent serum needs definite proof before its value can be accepted. Hyoscine is of great value as a symptomatic measure; scopolamine also is to be recommended. Surgical measures, such as cervical sympathectomy, have been used in a few cases with fair results. The Commission cannot recommend auto-serum therapy, the colloidal metals, iodides, malaria inoculations, non-specific protein therapy, salicylates, urotropine, and finds that the evidence is inconclusive in regard to the use of relapsing fever, sodium preparations, sulphur and vaccines.

There is little additional evidence produced, in the last year, with regard to the communicability of epidemic encephalitis, but there have been a number of additions to our knowledge of the epidemiology of the disease following the severe outbreak in Japan in 1929. The volume brings the bibliography of the disease to date and contains an excellent index. This publication is of first-class importance and demonstrates the value of a commission to study an individual disease in all its aspects.

Diabetes in Childhood and Adolescence. By PRISCILLA WHITE, M.D. Lea and Febiger. Philadelphia, 1932. Pp. 236. Price $3.75.

According to Dr. White, "The care of the diabetic child resolves itself mainly into three factors: (1) The maintenance of the normal physiological processes of the growing and developing organism; (2) the prevention of the accidents of diabetes; (3) the eventual production of an individual who will be an economic and a social asset." How these points may be realized is fully discussed in the various sections of the book.

There is brought out most strikingly the contrast between the diabetic child of the present day and that of the pre-insulin period. The various details of treatment, the methods of handling the manifold complications are clearly, concisely and accurately presented. Best of all, here is emphasized the practical disappearance of arteriosclerosis in adequately treated juvenile diabetics. Were this book to do nothing more than call attention to the possibility of the prevention of premature arteriosclerosis in the diabetic child and adolescent, the labor of its author would be amply repaid.

The illustrations are in keeping with the whole tone of the book. We see healthy diabetic children canoeing, on horseback, indistinguishable in appearance from their normal companions, affording concrete evidence that the objectives outlined by Dr. White are really capable of fulfilment.